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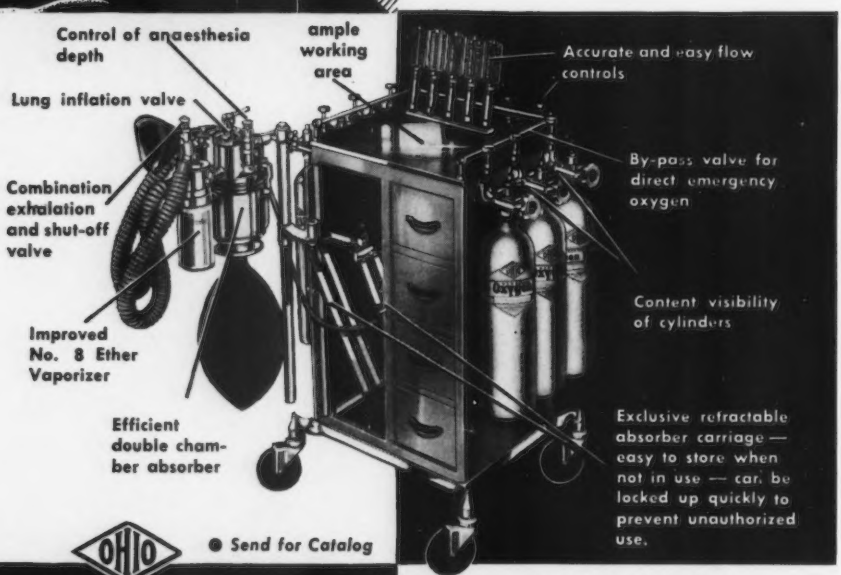
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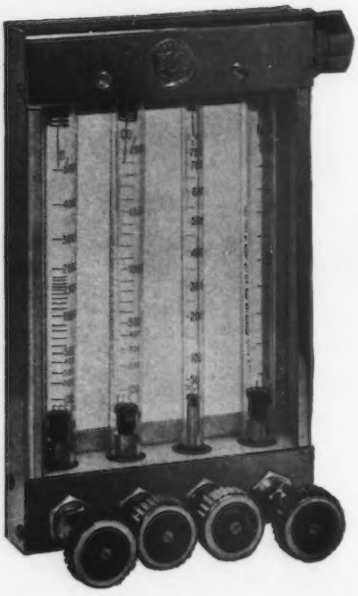

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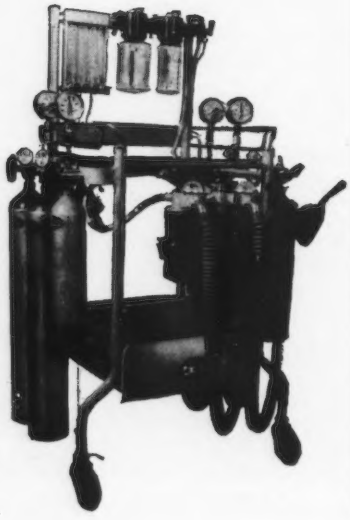

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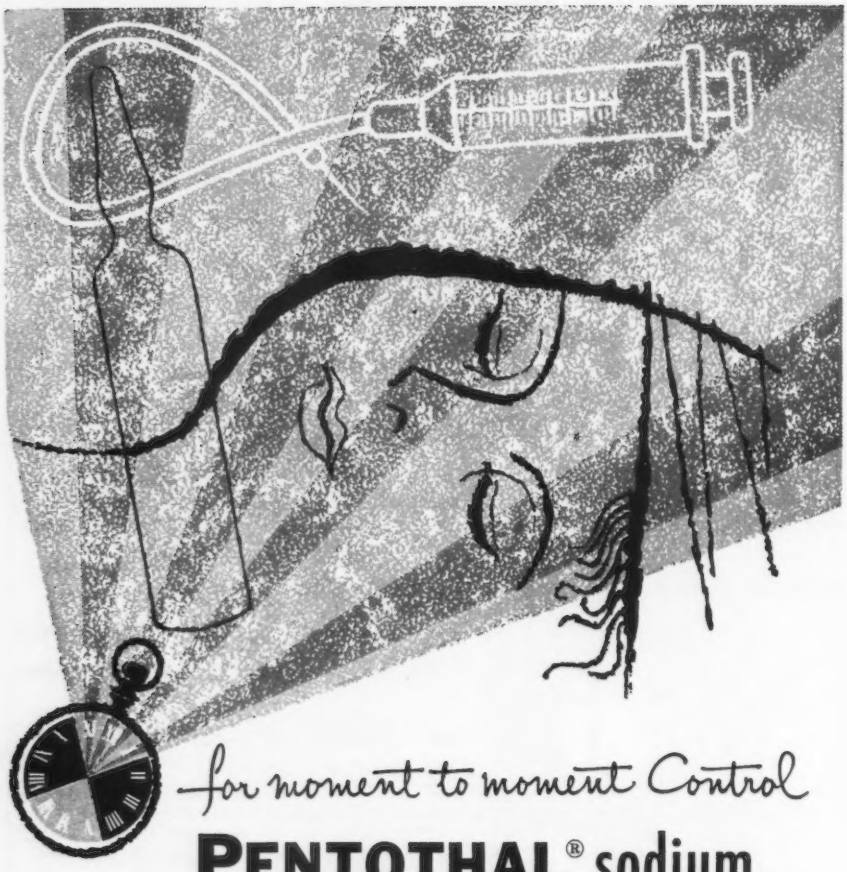

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EDITORIAL

We frequently hear the opinion expressed that there are "too many meetings." Undoubtedly this is true for those whose position in the profession entails the attendance at a great many medical meetings as contributors to the programme. It is doubtful, however, if it holds true for the practitioner who works alone or with a small group, and is unable to leave his practice at will or frequently, since in any community someone must remain at home to attend the sick; only when there are several possible choices during the year may everyone have the opportunity to attend a meeting.

The value of attendance at medical meetings is also questioned occasionally. For many practitioners in all branches of medicine such meetings provide the only possible postgraduate refresher courses. For others they provide an opportunity to compare experience with physicians from other centres, and to discover and in some measure evaluate new concepts and techniques which may be applicable in their own practice. Only by costly and extensive travel could such a wealth of information be gathered otherwise. Although it is true that most papers presented at the major medical meetings eventually find their way into print, yet these publications are not as valuable in this form as they are when they can be associated with the discussions which they evoke when presented at a meeting.

The combined meeting of the British Medical Association and the Canadian Medical Association in Toronto in June, 1955, will without doubt be of great value to all who are able to attend, no matter what their particular parochial interest in medicine may be. Difference of thought and custom are undoubtedly increased with distance, and the occasion of a meeting with members of the British Medical Profession must inevitably be stimulating and informative to both sides.

EDITORIAL

Nous entendons souvent dire autour de nous "qu'il y a trop de réunions". C'est sans doute un fait pour ceux que leur situation professionnelle contraint à assister à de nombreuses conférences médicales en tant que participants aux programmes. C'est plus douteux, cependant, pour le praticien qui travaille seul ou en petit groupe et ne peut à volonté quitter sa clientèle, du moins fréquemment, puisque dans toute communauté, il faut bien que quelqu'un reste à la maison pour soigner les malades; c'est seulement lorsque plusieurs choix s'offrent à lui au cours de l'année, qu'il peut avoir l'occasion d'assister à une conférence.

Vaut-il vraiment la peine d'assister aux conférences médicales? On en discute quelquefois. Pour beaucoup de praticiens dans toutes les branches de la médecine, ces conférences offrent au diplomé les seuls cours possibles pour se rafraîchir la mémoire. A d'autres, ils fournissent une occasion de comparer leur expérience à celle des médecins en provenance d'autres centres, de découvrir et dans une certaine mesure d'évaluer de nouvelles conceptions et techniques susceptibles de recevoir une application dans leur propre pratique. Ce n'est que par

de longs et coûteux voyages qu'ils pourraient acquérir d'une autre manière une telle somme d'informations. Il est sans doute vrai que la plupart des exposés présentés aux grandes réunions médicales sont tôt ou tard imprimés. Mais la valeur de ces travaux publiés est inférieure à celle qu'ils ont lorsqu'ils sont accompagnés des discussions qu'ils suscitent lors de leur présentation à un congrès.

Le Congrés mixte de l'Association Médicale Britannique et de l'Association Médicale Canadienne à Toronto en juin 1955 sera sans aucun doute d'un très grand intérêt pour tous ceux qui pourront y assister, quel que soit leur clocher médical. Les différences de pensées et de coutumes s'accroissent sans aucun doute avec les distances et l'occasion d'une rencontre avec les membres de la profession médicale Britannique doit inévitablement être stimulante et fructueuse pour les deux parties.

EXPERIMENTS WITH ANIMALS ON THE COMBINED ACTION OF PROCAINE AND BARBITURATES

MADELAINE O. MAYKUT, M.A.,* and WERNER KALOW, M.D.†

INTRODUCTION

Two principal effects of procaine on the central nervous system are known: excitant effects as manifested by convulsions, and depressant actions. Of the depressant actions, respiratory paralysis was the most obvious (1). A central analgesic action was then described (2, 3). More recently a hypnotic or anaesthetic effect in humans was observed. It was found that injections of procaine during anaesthesia would reduce the need for anaesthetic agents like thiopental, cyclopropane, or nitrous oxide (4, 5). Allen, Crossman and Lyons (6) and Wright (7) have used procaine as the main general anaesthetic agent with some premedication with barbiturate.

The clinical studies suggested synergism between procaine and barbiturates with respect to anaesthetic or hypnotic action. On the other hand, it is well known that barbiturates counteract the convulsions due to procaine, that is, in this respect the drugs are antagonists (8, 9). It was of interest to us to survey a system where both antagonistic and synergistic trends are present.

MATERIALS AND METHODS

All figures on doses and concentrations in this paper refer to the salts procaine hydrochloride, sodium pentobarbital (Nembutal), and sodium thiopental (Pentothal).

The experimental animals were white guinea-pigs of both sexes, with an average weight of 574 $\pm$13 gm. Some rabbits weighing between 2 and 4 kg. were used in addition.

Four series of experiments were performed on guinea-pigs. Common to these experiments was the following routine. The hair was clipped over the areas where injections were to be made. Sodium pentobarbital in 0.5 per cent solution was injected intramuscularly into both thighs of the animal. Immediately afterwards, procaine in a 2 per cent solution was injected subcutaneously on both sides of the back. When only one drug was given, physiological saline was injected instead of the other drug.

The first series of experiments was designed to survey broadly the effects of the combination of procaine and pentobarbital. Three guinea-pigs were tested at each dose level. Each animal was closely observed and notes were taken. The most conspicuous and consistent symptoms were subsequently translated to symbols explained in Fig. 1.

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†From the Department of Pharmacology, University of Toronto.

PENTOBARBITAL

PROCAINE mgm. per kgm.	0	5	10	20	40
0		OUU	UUU	SSS	SSS
50	OOO	OUU	UUU	SSS	SSS
100	EEE	UUU	UUS	SSS	SSD
200	UUU	UUS	UUS	SSS	SSS
400	CCC	CCS	SSS	SSS	SSD

O = no effect
E = excitement
C = convulsion
U = unsteady gait
S = sleep, narcosis
D = death

FIG. 1. Symptoms of individual guinea-pigs given various dose combinations of pentobarbital (Nembutal) and procaine.

In the next experimental series, "sleeping times" were measured. These were defined as the times of absence of the righting reflex. Seven determinations were made at each dose level.

The acute toxicities of pentobarbital and procaine, alone and in combination, were determined in the third series of experiments. All deaths occurred within six hours after the injections. The LD50's and their standard errors were estimated as described by Finney (10).

In experiments dealing with re-induction of sleep, procaine was injected not immediately after pentobarbital but at the first attempt of the guinea-pigs to move about while the effect of the barbiturate was ceasing.

The re-induction of sleep was also tested in rabbits using thiopental and procaine, both in 1 per cent solution, and both injected intravenously. An ear vein was cannulated with fine plastic tubing so that injections could be made without restraining the animal. Thiopental was injected to induce sleep for ten to fifteen minutes. The tube was then flushed with saline. The injection of procaine was started very slowly as soon as the righting reflex reappeared and was continued at a slightly increased rate until the rabbit was on its side again.

RESULTS

The most prominent symptoms of individual animals after injection of pentobarbital and procaine are shown in Fig. 1. Convulsions due to the highest dose of procaine alone are indicated in the lower left-hand corner of the diagram. Even small doses of pentobarbital suppressed this incidence of convulsions. Sodium pentobarbital, 10 mg./kg., used alone did not produce sleep but by the addition of higher doses of procaine sleep was induced. The last column of the diagram records two deaths when procaine and pentobarbital were combined. The results of this survey allowed the selection of suitable dose combinations for further studies.

At first the hypnotic action of the combined drugs was investigated. It was found that for comparison of different dose levels the duration of sleep was easier to measure than the depth of sleep. The doses used for sodium pentobarbital were 15, 21.3, and 30 mg./kg. and for procaine 100, 200, and 400 mg./kg. Seven guinea-pigs were used for each dose combination. All animals slept and none died. The results of these experiments are presented in the three-dimensional diagram (Fig. 2). The increasing doses of procaine are plotted on one axis from

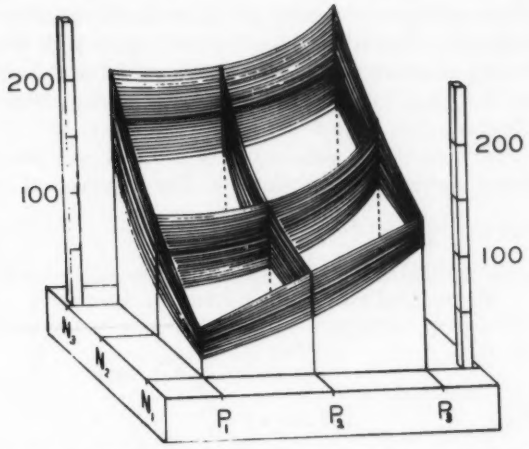

FIG. 2. Hypnotic action of combinations of procaine and pentobarbital (Nembutal) in guinea-pigs.

The two horizontal axes represent increasing doses of the two drugs; P stands for procaine, N for pentobarbital. The vertical axis represents sleeping time in minutes. The fence-like structure denotes the standard error of the mean sleeping time.

P1 to P3 and those of pentobarbital along the other axis from N1 to N3. The duration of sleep in minutes is represented by the height of the figure indicated by the two yardsticks. The average sleeping time is shown by the heavy lines joining the different dose combinations. The fence-like structure enclosing these lines represents the standard error of the mean. The lowest dose combination of pentobarbital and procaine (N1P1) gave rise to the sleeping time of about 30 minutes; the highest dose combination (N3P3) to 210 minutes. The surface of the diagram is curved. The fence-like structure is low for the low dose of pentobarbital and increases with increasing doses of pentobarbital. Although procaine also increases the sleeping time, the standard error does not increase with the dose of procaine.

The data were subjected to statistical analysis. Both procaine and pentobarbital have a significant influence on the sleeping time. There is also a significant increase of the error with increasing dose of pentobarbital, while the error does not change with increasing doses of procaine (11).

Another set of experiments was made to determine whether sleep could be

re-induced in guinea-pigs by the injection of procaine after the effects of pentobarbital had ceased. The sleep after the intramuscular injection of 30 mg./kg. of sodium pentobarbital lasted approximately one and a half hours. A dose of 200 mg./kg. procaine re-induced sleep in 3 out of 5 animals; 300 mg./kg. in 5 out of 5 animals. The re-induced sleep lasted approximately 100 minutes in the latter group. The sleep after the procaine injection was not so deep as that after pentobarbital injection. The bodies of the guinea-pigs appeared relaxed upon lifting them. However, an incomplete degree of relaxation was evident from occasional movements such as rolling over during the sleep. In experiments using rabbits, 5 out of 5 animals were forced back on to their sides with procaine injected intravenously during recovery from narcosis due to thiopental. The amounts of thiopental were 15-20 mg./kg., of procaine 12-16 mg./kg. Muscular relaxation could not be achieved.

Data on the acute toxicities in guinea-pigs of procaine and pentobarbital alone and in combination are presented in Table I. The influence of procaine on the

TABLE I

ACUTE TOXICITIES OF PENTOBARBITAL AND PROCAINE, ALONE AND IN COMBINATION
Pentobarbital, intramuscular: Procaine, subcutaneous

	LD50±S.E. mg./kg.	Slope*	Number of guinea-pigs
Pentobarbital Sodium	70.3± 4.7	7.9	35
Pentobarbital Sodium +300 gm./kg. Procaine	42.5± 4.3	5.4	35
Procaine	438 ±23	10.4	30
Procaine +10 mg./kg. Pentobarbital Sodium	674 ±18	22.8	30

*Probit units per log dose.

LD50 of pentobarbital, as well as of pentobarbital on the LD50 of procaine, is significant. If 70.3 mg./kg. is the LD50 of pentobarbital, 42.5 mg./kg. corresponds to the LD4, while the lethal effect of 10 mg./kg. is negligible ($<LD10^{-6}$). If 438 mg./kg. is the LD50 of procaine, 674 mg./kg. corresponds to the LD97, while 300 mg./kg. represents the LD4.

DISCUSSION

Our investigations on the duration of sleep have shown that procaine, capable of producing convulsions, is also capable of increasing the central depression due to pentobarbital. This could have one of two meanings: procaine either inhibits the elimination of pentobarbital or has a central depressing action. The experiments on the re-induction of sleep and the toxicity studies seem to favour the latter assumption. That is, the central depression due to procaine becomes

particularly evident if the convulsive component is suppressed by the addition of barbiturates. This conclusion supports previous observations by a number of different authors who claimed a central analgesic and depressant effect of procaine (2, 3, 4, 6, 7, 12, 13). The doses of procaine as well as of pentobarbital which were found effective in this study appear to be comparable to those used clinically if body surface is made the basis of comparison.

The relation between toxic and hypnotic effects of the two drugs is summarized in Fig. 3. The dose of pentobarbital is presented on one axis, the dose of procaine on the other. The lines in the diagram indicate equally effective dose combinations. The upper curve represents our data on the LD50's, the three lower curves are derived from observed and interpolated data which were presented in the three-dimensional diagram of Fig. 2. In such a plot as Fig. 3, a strictly additive effect is characterized by a straight line connecting both axes.

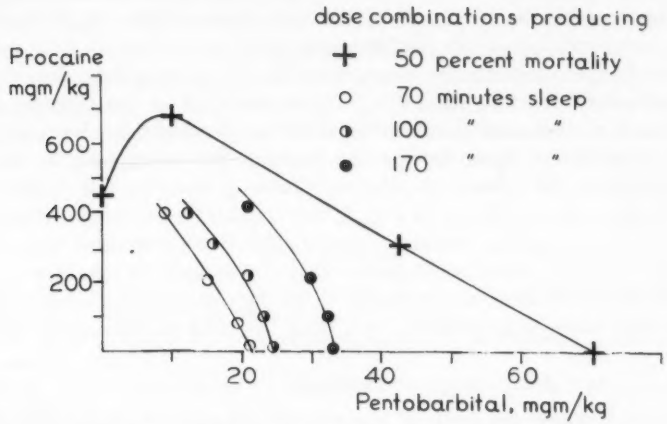

FIG. 3. Summary of data on the combined effects of pentobarbital (Nembutal) and procaine.
Upper curve: LD50's of pentobarbital and procaine, alone and in combination.
Lower curve: Dose combinations producing the same hypnotic action (cf. Gaddum (1940) Fig. 65, i.e. Fig. 76 in 4th ed., 1953).

The hump of the upper curve indicates the well-known antagonism of pentobarbital to the toxicity of procaine. Considering the experimental point at 42 mg./kg. of pentobarbital, by combining an LD4 of procaine and an LD4 of pentobarbital, we obtained 50 per cent mortality. The LD4 of pentobarbital is 42 mg./kg. Twice the LD4 of pentobarbital (84 mg./kg.) should kill 74 per cent, as indicated by the probit analysis. Thus, if procaine and pentobarbital could replace each other for their lethal actions, our drug combination should have killed 74 per cent instead of the observed 50 per cent. That is, the effect of 300 mg./kg. procaine is slightly less than additive to that of 40 mg./kg. pentobarbital. Loewe (14) classifies such an effort as a type of antagonism, Gaddum (15) as a type of synergism. Loewe's designation would give the intellectual satisfaction that the two drugs are called antagonists under all circumstances. The disadvantage of

this classification, however, is obvious. A patient under toxic influence of procaine may be saved by an administration of pentobarbital; a case of barbiturate poisoning could become fatal with an additional injection of procaine.

We interpret the combined action as follows. Procaine has at least two independent lethal actions on the central nervous system. One is counteracted by pentobarbital. The other appears to be augmentative to and perhaps identical in mechanism with the lethal action of pentobarbital. A similar mode of depressant action is suggested by the linearity of the major portion of the mortality curve of Fig. 3.

An extrapolation of the three lower curves (Fig. 3) towards the ordinate would give us theoretical values for the hypnotic activity of procaine. This action of procaine is probably not completely additive to that of pentobarbital. The extrapolated portions of the three curves would intersect the mortality curve. This means, first, that the hypnotic effect of procaine cannot be ordinarily demonstrated because of the existence of the convulsive action; secondly, it again suggests independence of the two effects of procaine.

The course of the mortality curve and the sleeping curves shows that the margin of safety (that is, the ratio of the toxic dose to the effective dose) is diminished if we enhance the narcotic action of pentobarbital by procaine. For a range of doses this disadvantage may perhaps be outweighed by the greater predictability of the actions of procaine. Table I indicates the steep mortality curves of procaine. As shown in Fig. 2, the standard error of the sleeping time increased with increasing doses of pentobarbital but remained constant with increasing doses of procaine. It means that if we want to produce sleep of a given duration, we have a better chance to hit the right time by combining pentobarbital with procaine in proper doses, than by using pentobarbital alone.

Summary

Procaine prolonged the sleep of guinea-pigs induced by pentobarbital.

Procaine could re-induce sleep in guinea-pigs and rabbits after the apparent effects of pentobarbital or thiopental respectively had ceased. This is evidence of an hypnotic effect of procaine rather than of an influence on the elimination of the barbiturate. The hypnotic and the convulsive effects seem to represent two independent actions of procaine.

The effect of procaine on the toxicity of pentobarbital was nearly additive. The effect of pentobarbital on the toxicity of procaine was antagonistic.

The distribution of standard errors of the sleeping times allowed the conclusion that the duration of the sleep could be better predicted if induced by the combination of pentobarbital and procaine than by pentobarbital alone. However, we appeared to lose on the margin of safety with this combination of drugs.

Acknowledgements

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Epidemiology and Biometrics for their efforts to analyse an unusual statistical problem.

Résumé

Le synergisme autant que l'antagonisme ont été signalés dans les écrits sur l'action combinée de la procaine et des barbituriques. En conséquence, on a entrepris une étude expérimentale sur l'influence réciproque des deux drogues. La procaine prolonge chez les cobayes l'anesthésie initiée par le pentobarbital. La procaine a pu provoquer à nouveau le sommeil chez les cobayes et les lapins après que les effets apparents du pentobarbital ou du thiopental eussent respectivement cessé. Cela met en évidence un effet hypnotique de la procaine plutôt qu'une influence sur l'élimination des barbituriques. La toxicité aigüe des deux drogues, seules ou en combinaison, a été mesurée en déterminant la dose mortelle (LD50) chez les cobayes. L'effet du pentobarbital (acide diethyl-pentobarbiturique) sur la toxicité de la procaine a été antagoniste. L'effet de la procaine sur la toxicité du penobarbital, cependant, a été presque additif.

Les effets hypnotiques et les effets convulsifs paraissent représenter deux actions indépendantes de la procaine. La distribution des erreurs normales permet de prévoir la durée du sommeil relativement mieux avec la combinaison qu'avec le pentobarbital seul. Cependant, cet avantage semble être contrebalancé, puisqu'on perd sur la marge de sécurité avec cette combinaison de drogues.

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NEUROLOGICAL COMPLICATIONS OF SPINAL ANAESTHESIA*

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A GREAT DEAL has been written in the literature and presented in discussions at meetings concerning the neurological complications of spinal anaesthesia. Many investigations of clinical and experimental nature have been undertaken in order to discover the true perspective in the incidence of morbidity and mortality. The basis of this debate lies in the incidence and severity of the neurological complications. These may be described under the headings of Immediate and Subsequent.

The *immediate* neurological complications concern the excessive height to which the anaesthetic solution might rise, thereby producing undesired paralysis of nerves and nerve centres. This complication is mentioned as it has produced a fatal outcome in the hands of the unalert. It has been known to cause a worse result, namely irreversible anoxic changes in the brain in patients who have otherwise recovered. Amongst 77 fatal cases of cerebral anoxia studied by Courville (1) no less than 12 were due to spinal anaesthesia.

The *subsequent* possible complications are known to all; indeed some have been in the unpleasant position of being associated with one or more of the serious types.

POST-SPINAL HEADACHE

Etiology

It is now well established that the common type of post-spinal headache is caused by leakage of cerebrospinal fluid which creates low pressure within this system. The altered dynamics cause "sagging" of the architecture of the brain, the consequent tension on the sensitive parts of the brain producing this headache. McNaughton (2) has shown that the sensory nerve supply of structures above the tentorium is the fifth while the posterior fossa is supplied by the ninth, tenth and upper cervical nerves.

Kreuger (3) quotes Sicard for having put forward the suggestion of leakage in 1902. This theory has been followed by others (4, 5, 6). Thorsen (7) points out that with 84 per cent of all post-spinal headaches there is hypotension of the cerebrospinal fluid.

Kunkle (8) and others have shown that the loss of 20 ml. of cerebrospinal fluid is enough to lower the intracranial pressure; this fall is more marked when the posture is erect.

Additional Factors

Other causative factors have been well put forth by Kreuger, Stoetling and Graff (9) who describe the importance of dehydration, trauma, size of needle

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and apprehension, in the incidence of post-spinal headaches. The actual anaesthetic agent introduced does not appear to be of statistical importance; Bonica (10) states that the addition of vasoconstrictors to the solution bears no relationship to this occurrence.

As well as the factors enumerated above, McCord (11) and others feel that cervical cord pathology may be of significance and that meningeal irritation should be considered. This latter item raises a different etiological factor which will be mentioned later.

TABLE I

INCIDENCE OF POST-SPINAL HEADACHE

Investigation	No. of cases	Details	Frequency
Brady (13)	1148	No. 22 needle Cont. Spinal No. 3½ catheter	3.9% 6%
McCord (11) *et al.*		Vag. delivery	14–18%
(Cases quoted)		Various series	1–32.8%
Kreuger (3)		"Ordinary needle" No hydration	21.7%
Kruger (3)		Average all types	10.8%
Kreuger (3)		Whitacre needle	4%
Kreuger (3)		Apprehensive patients	14.3%
McCarthy (14)		Over-all	15%
Harris (15)		No. $\frac{20}{24}$ needle	$\frac{2}{1}$
		Catheter technique	23.8%
Gilbert (15)	912	Over-all	2.5%

Many of the quoted authorities point out the higher incidence of headaches in obstetrical cases and are agreed concerning the reasons for this fact: relative dehydration, early sitting up, and the effects of pressure changes due to the abdominal tumour. To these may be added the dissimilarity to surgical patients who usually lie longer in bed and receive more anodynes than does the average obstetrical charge.

For a comprehensive chart concerning the incidence of headache, showing larger numbers of cases, reference may be made to that produced by Pitkin (12).

Prophylaxis

Kreuger (3) has quoted Antoni as having described the importance of a small needle in 1923. Associated with this has been the assertion that a needle should separate the fibres of the arachnoid rather than cut them; hence other modifications have been described in the form of the needle. By these means the finest of holes is made which may readily be sealed off.

Green (17) and others have stressed the importance of preventing a headache by efficient hydration and by making use of the antidiuretic property of posterior pituitary extract as well as of a small gauge needle.

Kaplan (18) has suggested and carried out the procedure whereby 10–20 ml. normal saline is introduced (epidurally) as the spinal needle is withdrawn.

Treatment

Nursing in the supine position and treatment by aspirin combinations have, in the past, cured the majority of headaches. Are there other measures which should be undertaken in the early stages? What should be done in the case of severe persistent headaches? These are questions which should be considered if spinal anaesthesia is still to be used.

Weed and McKibben (19) described the changes which occurred in the pressure of cerebrospinal fluid following intravenous injections of varying concentration. This concept has lead to the intravenous administration of 5 per cent dextrose or 2.5 per cent dextrose in .45 per cent saline. To this solution has been added 100 mgm. nicotinic acid (3).

Brady (13) recommends Empirin and nicotinic acid. McCarthy (14) advises caffeine sodium benzoate. McCord (11), Ahearn (6), Wolff (4) and Rice (20) all advocate, as do many others, the intrathecal or epidural injectine of saline: 70–100 ml., given to treat a severe headache, will cure all but 20 per cent. This group will respond to a second treatment. A modification of this has been advocated at the Montreal Neurological Institute when an artificial C.S.F. produced by Dr. K. A. C. Elliott is used intrathecally. This has a physiological rationale to recommend it (21). Glesne (22) recommends the use of intrathecal glucose, with which 50 per cent success is claimed.

Deutsch (23) reports the treatment of severe post-spinal headache by intravenous ethanol. One litre of 5 per cent alcohol in 5 per cent glucose is given. This appears a rational form of treatment. The alcohol is an analgesic and provides euphoria, it gives rise to dilatation of pial vessels (24), it aids sodium retention (25) and causes a shift of water from the intracellular to the extracellular compartment (26).

Other types of headache

There remain a small percentage of headaches which are not of the previously described type. They are usually associated with a high cerebrospinal fluid pressure and a high C.S.F. cell count. The symptom usually suggests meningismus rather than the frontal or occipital headache of the low pressure type of syndrome. This type of headache is not relieved by posture. The occurrence of this pain calls for close observation and investigation, following which specific treatment may be indicated. Apart from this, treatment is symptomatic.

Cranial Nerve Lesions

These may occur following spinal anaesthesia owing to different etiological factors. First, and most commonly, they may be produced by alterations in the architecture of structures within the skull brought about by altered hydrodynamics whereby the nerves become stretched. Secondly, they may be brought on by a meningitic phenomenon either septic or aseptic. Thirdly, they may be the outcome of widespread adhesive changes such as will be described later. Kennedy and Lockhart (27) have enlarged upon these etiological factors.

The first and common type, that of the sixth nerve, occurs with varying frequency. Some report large series with scarcely a case while others report its occurrence with apparent frequency. The fortunate fact concerning this diplopia-producing complication is that it nearly always rectifies itself in time. For this reason there should be no undue haste in seeking ophthalmic surgeons to perform muscle operations.

Though of much rarer occurrence, paralysis of the other cranial nerves has been reported (7).

Other Nerve Palsies

Isolated nerve palsies, usually reversible, may occur in relation to the cauda equina—a typical example is that which gives rise to foot drop. Other sensory or motor nerves may be temporarily or more rarely, permanently, involved.

The other types are of much graver consequence and are symptoms from other clinical pictures.

Arachnoiditis, Radiculitis, Neuritis, Myelitis, Cauda Equina Syndrome and the Neurogenic Bladder

These form a group of complications of the utmost severity. They are far more grave than those complications which might be expected to follow general anaesthesia. It is complications as these especially which have given rise to such controversy (28, 7, 29). Two such cases have been admitted to the Montreal Neurological Institute during the last four years.

Pathology

These cases may present a variety of lesions, some or all of which are found. By the time some cases reach autopsy, fibrosis and degeneration are the most prominent features.

Nerve roots present early degeneration of the medullated fibres followed by complete degeneration.

The dura, arachnoid and pia become thickened and adherent. Eventually they fuse one with the other.

Myelitis of static or ascending nature may be found.

In some cases there has been reported involvement of meninges and cranial nerves, also evidence of encephalitis.

A case was seen recently (30) in which the patient never recovered from the effects of a spinal anaesthetic with 1/1500 Nupercaine given for gastrectomy. A standard technique was used. Following this procedure myelography determined

a spinal block from T_5–L_2. Laminectomy was performed within a few days. At this operation, the portion of the cord involved appeared grossly congested and swollen. The appearance suggested infarction. The decompression has not yet resulted in recovery from this paraplegia.

Investigations

McCarthy (14) quotes but gives no reference to Davies as having produced experimentally changes in the meninges, ganglion cells and nerve fibres which are similar to changes found in cases which have had fatal outcome, owing to neurological damage.

It has been considered that in humans a number of cases may develop early but reversible changes. This is an unknown factor.

Sterilization solutions and green soap have been injected in larger than clinical doses into the subarachnoid space of dogs (29); all have recovered.

Clinical Picture

The onset of symptoms may be immediate or of gradual development during the course of days or weeks. The progress may become arrested, resulting in partial or complete paraplegia. The condition more commonly, however, progresses, either rapidly or slowly, to a fatal termination.

Causation

There are a number of possible causes:

1. The *drug* itself may have specific affinity for a specific spinal cord or it may be in a too high concentration in a particular case. In either of these cases an irritant process is initiated leading to reversible or irreversible changes.
2. There may be direct injury to nerve, cord or blood supply. The conus medullaris may be injured by too high a puncture. A recent case may be cited (31) in which a cauda equina syndrome followed rapidly a spinal anaesthetic. The site of lumbar puncture could still be seen, a silver clip was fixed thereon and a film was taken. The X-ray showed the puncture was made between T_{12} and L_1.
3. There may be chemical contamination of the anaesthetic solution injected.
4. There may be a low-grade infection, either exogenous or endogenous.
5. There may be coexisting disease of the central nervous system.

Concerning the latter possibility, a personal case can be mentioned in which a patient, who had previously suffered from infantile paralysis, was anaesthetized for an operation on the knee joint. Subsequently, that patient appeared to have paraplegia. The patient was removed to the neurological department of another hospital with the diagnosis, spinal complication. In actual fact, the patient had received a general anaesthetic, as her chart bore witness. The diagnosis was then altered to one of hysteria.

Less Common Complications

Meningitis

Little need be said concerning this dreadful and entirely avoidable complication.

Paravertebral Block

A paravertebral block with pontocaine has been reported (32) as causing radiculitis. Efocaine injected paravertebrally (33, 34) has caused paraplegia.

Herniated Intervertebral Disc

This is not a common sequela but there is convincing evidence that undue trauma at lumbar puncture may, by piercing the annulus fibrosis, precipitate the herniation of a Nucleus Pulposus. It may be that that disc is already the site of degenerative changes.

Pros and Cons of Spinal Anaesthesia

There are ardent protaganists of spinal anaesthesia such as Cole (35). There are other men who cannot condemn the method too strongly.

The arena is also entered by our surgical and obstetrical colleagues, some of whom maintain that they will only operate under spinal anaesthesia while others refuse to allow their patients to be subjected to such a form of anaesthesia.

If spinal anaesthesia is to be used, the greatest care must be exercised in the preparation of the equipment; infinite dexterity and sterility must govern the procedure; prophylactic measures should be taken to ensure a minimum of even the more minor complications.

My opinion remains the same as when written in 1952 (36): "In spite of the crippling conditions inevitably seen at the big Neurological Centres, the general impression remains that these conditions are in reality rare and there is no need to abandon a sound form of anaesthesia WHEN INDICATIONS ARISE FOR ITS USE."

Résumé

On a beaucoup écrit et discuté dans les conférences médicales sur les complications neurologiques de la rachi-anesthésie. La base de ces discussions réside dans l'incidence et la gravité de ces complications.

Les complications neurologiques immédiates sont en relation avec une élévation trop marquée de la solution anesthésique, qui produit une paralysie imprévue des nerfs et centres nerveux. Cette complication peut entrainer la mort ou des altérations irréversibles du cerveau dues à l'anoxie.

Parmi les complications ultérieures de la rachi-anesthésie, les céphalées sont les plus courantes. On a bien établi aujourd'hui que le type courant de céphalée post-rachi-anesthésique est provoqué par une perte du liquide céphalo-rachidien, qui déclenche une baisse de pression à l'intérieur du système. D'autres facteurs importants sont la déshydratation, les traumatismes, la dimension de l'aiguille et l'appréhension. L'agent anesthésique lui-même ne paraît pas revêtir d'importance statistique. Pour prévenir l'apparition de céphalées, on recommande les mesures suivantes: emploi d'une petite aiguille, hydratation efficace, emploi des propriétés anti-diurétiques de l'extrait post-hypophysaire, et injection de soluté physiologique normal dans l'espace épidural quand on retire l'aiguille.

Dans le passé, on a pallié à la plupart des céphalées consécutives à la rachi-anesthésie en couchant le malade en position dorsale, et par l'administration

de composés d'aspirine. Les céphalées plus sérieuses et persistantes ont été traitées par des injections intraveineuses d'une solution à 5 pour cent de dextrose, pouvant contenir de l'acide nicotinique; par l'"Empirine" et l'acide nicotinique; par la caféine et le benzoate de sodium; par des injections intra-rachidiennes ou épidurales de soluté physiologique normal (70–100 ml) ou de liquide céphalo-rachidien artificiel ou de solution de glucose; et par des injections intraveineuses d'éthanol.

D'autres types de céphalées post-rachi-anesthésiques peuvent être associées à des signes méningés, à une hypotension et à une présence élevée de cellules dans le liquide céphalo-rachidien.

Les lésions des nerfs craniens qui suivent une rachi-anesthésie peuvent être dues à une altération des réactions intra-cervicales ou à un phénomène méningé septique ou aseptique. Le nerf le plus couramment affecté est le sixième nerf cranien. On a signalé la paralysie d'autres nerfs craniens. Heureusement cette complication se résout toujours d'elle-même avec le temps.

L'Arachnoidite, la névrite radiculaire, la Névrite, la Myélite, le syndrôme Cauda Equina et la Vessie neurogène composent un groupe de complications extrèmement sérieuses. L'apparition des symptômes peut être immédiate ou se développer peu à peu en quelques jours ou en quelques semaines. Parmi les causes possibles, citons:

1. L'irritation due à l'agent anesthésique rachidien lui-même
2. La lésion directe d'un nerf, de la moelle épinière, ou des vaisseaux sanguins
3. La contamination chimique de la solution anesthésique
4. L'infection mineure, exogène ou endogène
5. La présence simultanée d'une maladie du système nerveux central

L'auteur est d'avis "qu'en dépit des infirmités que l'on rencontre inévitablement dans les grands centres neurologiques, l'impression générale demeure que ces cas sont en réalité rares et qu'ils ne doivent pas entraîner l'abandon d'une forme d'anesthésie qui reste valable *quand son emploi est indiqué.*"

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A STUDY OF BODY TEMPERATURE DURING GENERAL ANAESTHESIA*

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ALTHOUGH the vernacular phrase "out cold" was in use to describe the state of anaesthesia well before 1885, not until that year was clinical evidence presented to verify this homely observation, when Kappeler (1) noted that during narcosis produced by ether or chloroform a patient's body temperature might fall as much as 1.5° C. in one hour. Hare (2) found that even greater losses occurred in the axillary temperature during etherization and in dogs he observed decreases of 8–10° F. rectal temperature during an hour. Allen (3) confirmed the tendency of the etherized dog to become hypothermic at room temperatures of 65–75° F. (18–24° C.) but he also demonstrated that the fluctuation in temperature could be reversed by the application of external heat or at a room temperature of 93–96° F. (34–36° C.). Similar results with dogs were obtained by Mills (4). Davis (5) in a series of animal experiments showed that the use of wet towels aggravated the heat loss, but that if the ether vapour was warmed the rectal temperature would remain stationary, even at a room temperature of 21° C. He observed during 165 surgical anaesthetizations by the open drop ether technique that the rectal temperature fell less if the patient was covered and dry, than if he was exposed and moist. In addition, he found that as the operating room grew warmer, the average loss decreased from 0.75° C. at 24.0° C. to only 0.07° C. at 32.5° C. room temperature.

That the etherized dog actually becomes poikilothermic was conclusively demonstrated by Barbour and Bourne (6) in 1923; they showed that normal dogs under ether anaesthesia are so lacking in ability to regulate their body temperature that it remains constant only at an environmental temperature of 30.5° C. in either wet or dry atmospheres. In the child as well, homeothermic regulation is profoundly disturbed during anaesthesia; at operating room temperatures of only 76–82° F. (24–28° C.) Bigler and McQuiston (7) found that 62 per cent of anaesthetized children became hyperthermic and even cooling with ice bags or a water mattress from the start of the operation could not prevent increases in rectal temperature above 100° F. (38° C.) in 40 per cent of the cases. In adult man, the evidence for poikilothermia during anaesthesia has not been so conclusively demonstrated. Watkins and Wilson (8) found that although heat losses occurred at ambient temperatures below 70° F. (21° C.), even in long operations only relatively small decreases in rectal temperature were noted. Léger (9) observed maximum hypothermias of 1.5° C. and 0.8° C. for inhalation and spinal anaesthesia respectively. Post-operative heat stroke has been attributed to high operating room temperatures (10, 11) and at air temperatures

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above 85° F. (29° C.) the use of the closed soda-lime absorber technique has produced hyperthermias of 5–7° F. Although Searles and Lenahan (12) consider the anaesthetized patient to be poikilothermic and the room temperature the most important factor modifying rectal temperature, Smith (13) while investigating the aetiology of ether convulsions, measured the theatre air temperature, air velocity, relative humidity and temperature under the drapes of 62 operating cases and 22 controls and concluded that there was "absolutely no correlation between the theatre temperature and rectal temperature."

More recently Clark, Orkin and Rovenstine (14) inferred from a study of 164 adult patients that the wet bulb temperature is the most satisfactory measure of the thermal tolerability of the operating room environment. They found that between 58 and 75° F. (14 and 24° C.) wet bulb, rectal temperatures rose but not excessively and heat retention occurred only once in 119 individuals. Above 75° F. (24° C.) wet bulb, rectal temperatures increased 1.2–2° F. per hour and heat retention occurred in 9 cases out of 45. In both wet bulb ranges the closed to-and-fro rebreathing system was associated with greater temperature increases than either the closed circle rebreathing system or the non-rebreathing system.

The temperature of the skin during anaesthesia has also been studied. Foged (15) observed that both ether narcosis and spinal anaesthesia normally caused an average increase of 5° C. in the skin temperature of the foot. Others (16, 17, 18) have shown that the increase is as great as that following sympathectomy and that in the dog vasoconstriction in response to a sudden decrease in environmental temperature does not occur. Foregger (19) has demonstrated maximum increases in the cutaneous temperature of the great toe by inhalation of nitrous oxide, ethylene, cyclopropane, ether and chloroform, by intravenous pentothal, and by spinal procaine. The phenomenon occurred in the first stage of anaesthesia and was not associated with a fall in arterial blood pressure.

In some preliminary measurements in an air-conditioned operating suite, Scott (20) observed depressions of 4 and 5° C. in the rectal temperature of several neurosurgical patients. It was decided to investigate these findings more thoroughly in the present study and because the work was done during the summer months, to extend the observations to operating rooms which were not mechanically cooled, in an attempt to determine more accurately the factors regulating body temperature during anaesthesia.

Materials and Methods

The behaviour of the rectal, skin, and muscle temperature and of the sweat glands in 35 afebrile adults ranging in age from 17 to 72 years, undergoing general anaesthesia of at least one hour's duration, was studied. In each instance a description of the thermal environment in terms of dry and wet bulb temperatures and air velocity was obtained. None of the patients suffered shock and all survived the operative procedures.

Temperatures were measured with an "Ellab" thermometer type T.E. 3 (manufactured by Electrolaboriet, Copenhagen) which is a mirror galvanometer compensated for use at room temperatures of 10–31° C. and reading directly in degrees Centigrade from a variety of thermocouple applicators. Readings were

taken at six-minute intervals. An applicator consisting of a thermojunction within a bright metal plug was inserted 8–12 cm. into the rectum when anaesthesia was achieved. A bare wire thermocouple set in a plastic holder was attached to the surface of the lower limb with adhesive tape in such a way that the junction was left uncovered; a loop of wire containing a thermojunction was similarly applied to the great toe. A few readings of skin temperature were usually made before anaesthesia was induced. Muscle temperature was measured with a thermocouple set in the point of an injection-type needle which had a removable outer cover which could be sterilized. A thermocouple was suspended at the foot of the operating table to measure room air temperature (dry bulb) while a bare wire thermocouple set in a plastic rod was fixed beneath the surgical drapes 6–8 cm. from the lateral surface of the patient's thigh. The accuracy of the instrument was ascertained at ± 0.1° C. and all applicators used read within ±0.1° C. of each other. Although the accuracy of the surface temperature readings was not separately determined, others (21) have reported an accuracy of ±0.15° C. with similar devices.

The wet bulb temperature was measured at thirty-minute intervals with a sling psychrometer accurate to 0.5° F. or in some cases in the air-conditioned theatre the relative humidity was read from a wall hygrometer and the corresponding wet bulb value obtained from psychrometric tables. Air velocity was determined at a distance of 6 feet from the patient with a silvered kata thermometer (cooling range 130–125° F.).

The presence or absence of sweating was demonstrated by Minor's method (22): the skin of the dorsum of the foot, the calf or the face was painted with a solution of iodine (iodine 2 gm., castor oil 10 ml., absolute alcohol to 100 ml.) and dusted with dry rice starch.

The neurosurgical patients were anaesthetized in an anteroom and then transferred to the air-conditioned operating room. The others were anaesthetized in operating rooms which were ventilated by open windows or in one instance by fans. In addition, the neurosurgical draping procedure differed in that the drapes, by being carried over an instrument stand elevated above the centre of the operating table, were held away from the body and permitted access to the patient by the anaesthetist; in other surgical procedures the drapes fell directly upon the patient. Preoperative medication consisted of atropine 0.6 mg. or 0.4 mg. in all but one case, with morphine 10 mg. in 16 cases and Demerol 50–100 mg. in 9 cases.

Anaesthesia was induced in all patients with intravenous Pentothal Sodium in total dosages of 0.2–1.0 gm. Muscular relaxation was achieved in all cases by the intravenous administration of one or two of the following:

Succinylcholine chloride 20–80 mg. 19 cases;
D-tubocurarine chloride 9–22 mg. 3 cases;
Flaxedil 4–5 mg. 3 cases;
Decamethonium bromide 2–5 mg. 7 cases.

Anaesthesia was maintained by inhalation using a variety of anaesthetic agents. Three systems were used for administration: the closed circle rebreathing

system, the semi-closed partial rebreathing, and the non-rebreathing system. Gases were introduced by endotracheal intubation, by endopharyngeal airway with cup, or via a tracheotomy. Table I shows the various combinations of agents and techniques employed and their distribution among the cases studied. Some of the totals exceed 35, the total number of patients, because occasionally more than one technique was used during a single operation.

TABLE I

ANAESTHETIC AGENTS, BREATHING SYSTEMS, AND AIRWAYS USED

Breathing System	Non-rebreathing	Semi-closed	Closed	Totals
Number of cases	2	22	14	38
ANAESTHETIC AGENTS				
Cyclopropane	0	0	3	3
Ether	0	1	8	9
Ether-cyclopropane	0	0	1	1
Nitrous oxide (N_2O)	1	5	0	6
N_2O-Trilene	1	5	1	7
N_2O-ether-Trilene	0	4	0	4
N_2O-cyclopropane	0	1	0	1
N_2O-ether	0	6	1	7
AIRWAY				
Endotracheal Tube	1	17	11	29
Endopharyngeal Airway with Cup	1	4	2	7
Tracheotomy	0	1	0	1

Intravenous fluids were continuously administered to 31 patients during their anaesthesia. Of these, all received saline, 20 also received blood, and 3 dextran. The mean volume received was 1,000 cc. In 12 cases the blood was administered at refrigerator temperature (about 5° C.).

RESULTS

The extent and direction of the changes observed in the rectal temperatures are shown in Figs. 1, 2, and 3. For each of the three groups of environmental conditions the deviations from the value at the time of induction (0 hours) have been plotted at successive intervals of 18 minutes. A smooth curve joins the average change at each interval. The distribution into the three groups was made according to the mean ambient dry bulb temperature, calculated for each case. The wet bulb temperatures simultaneously observed have been indicated also.

The mean rectal temperature at induction was 37.2 ± 0.4° C. (S.E. of mean of 35 observations) and the range 36.2–38.0° C. Thereafter, with one exception mentioned below, no rectal temperature was observed beyond the limits 36.0–38.6° C. and in only 4 cases was 38.0° C. exceeded. The mean duration of

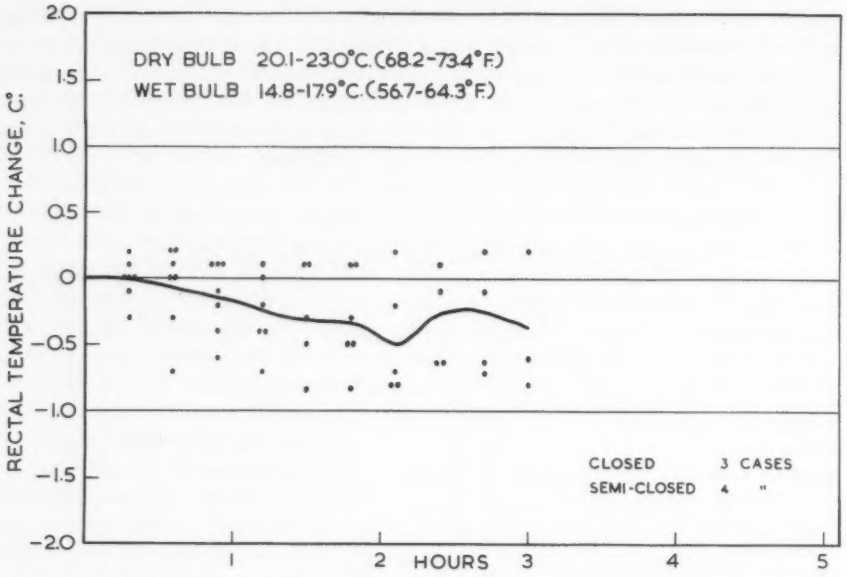

FIG. 1. Rectal temperature changes during anaesthesia at air temperature of 20.1–23.0° C. Time of induction is 0 hours.

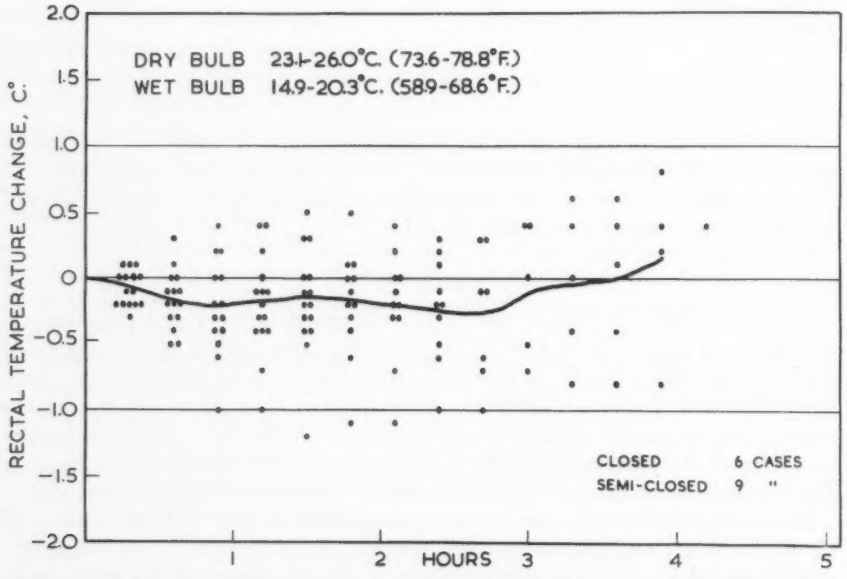

FIG. 2. Rectal temperature changes during anaesthesia at air temperature of 23.1–26.0° C. Induction at 0 hours.

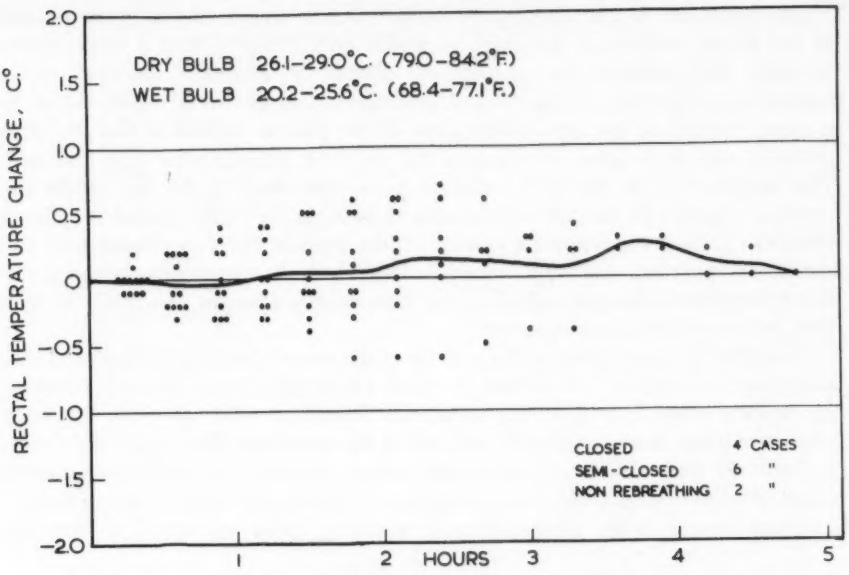

FIG. 3. Rectal temperature changes during anaesthesia at air temperature of 26.1–29.0° C. Induction at 0 hours.

anaesthesia was 2 hours 51 minutes ± 1 hour (35 obs.) with a range, 1 hour–5 hours 30 minutes.

One rectal temperature record was not included in the accompanying charts. In this case the mean dry bulb temperature of the room (19.0° C.) was the only value observed below 20.1° C. and although a marked change in rectal temperature occurred, no separate graph was drawn. This patient was anaesthetized through a tracheotomy tube with the semi-closed partial rebreathing system; his rectal temperature fell from an initial 37.3° C. to 34.1° C. during 4 hours of anaesthesia, although in the final hour the decline was less marked than earlier. No shivering was observed.

In 16 other cases in which the rectal temperature showed decreases of 0.3–1.2° C. shivering was noted in only one case. This patient had received 3 mg. decamethonium bromide as a muscular relaxant. The mean air temperature was 22.2° C. The shivering continued without interruption for 2 hours; the mean rectal temperature was constant at 37.1 ± 0.1° C., after having fallen 0.8° C. before shivering commenced.

The temperature of the quadratus femoris muscle was observed in 4 cases. The mean muscle temperatures were

$$0.9 \pm 0.1^\circ \text{ C.}, \quad 1.0 \pm 0.1^\circ \text{ C.}, \quad 1.4 \pm 0.2^\circ \text{ C.}, \quad \text{and} \quad 2.2 \pm 0.4^\circ \text{ C.}$$

below the respective rectal temperatures. In two cases the curves of rectal and muscle temperature were parallel, but in the other two cases they diverged with the progress of the operation.

The behaviour of the temperature of the plantar surface of the great toe and of the lateral surface of the thigh is described in Figs. 4 and 5 respectively. Absolute skin temperature readings are plotted at successive intervals of 18 minutes from the time of induction of anaesthesia and the mean values joined by a curve. Records of the skin temperature of the plantar surface of the heel in 5 patients, and of the medial surface of the calf in 6 patients were also obtained. The temperature of the heel behaved much like that of the toe, while the changes observed in the calf were similar to those in the thigh. Except at 0 hours when the patient was not yet unconscious, the level of the skin temperature and the air temperature were apparently independent. A comparison between the skin temperature changes and the rectal temperature changes was made in each case, but no relation was apparent.

A satisfactory description of the activity of the sweat glands in the selected skin areas was obtained for 14 patients. In cases where Minor's test showed sweating on exposed parts, unevaporated sweat was found on other parts of the body when the drapes were removed at the end of the operation. The results are shown in Table II and additional information about ambient dry bulb temperature, rectal temperature, and the administration of intravenous fluids is also given.

Air movement in the air-conditioned operating room was 49 ± 10 feet per

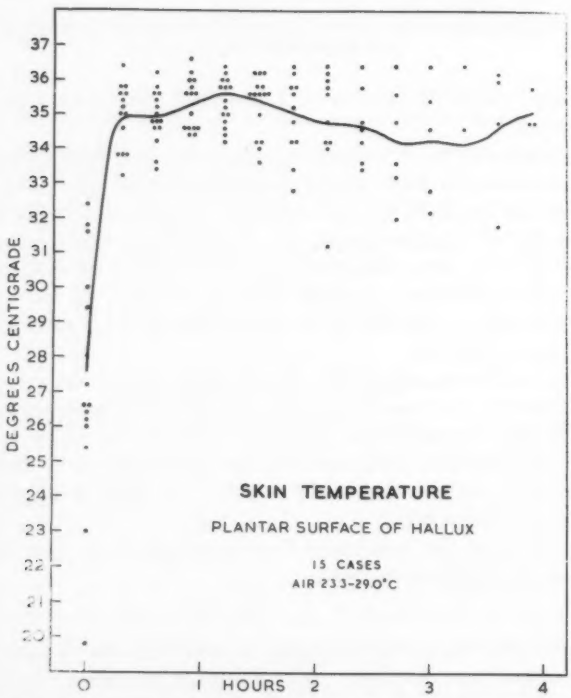

FIG. 4. Skin temperature of plantar surface of the great toe during anaesthesia.

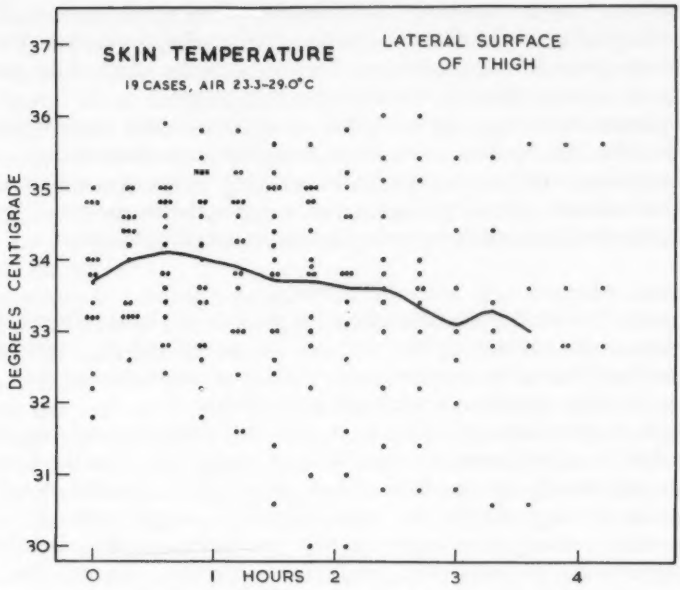

FIG. 5. Skin temperature of lateral surface of the thigh during anaesthesia.

TABLE II

SWEATING DURING ANAESTHESIA

	Case	Mean dry bulb temp., °C.	Intravenous fluid, c.c.	Max. variation in rectal temp., °C.
Sweating observed	La.	29.0	900	±0.2
	Ma.	28.8	800	+0.4
	Cr.	28.6	500	+0.3
	Kr.	28.0	1100	+0.4
	Lo.	27.0	0	+0.2
	Ba.	27.1	2500	−0.4+0.5
	v.B	26.8	1200	−0.6
	Hi.	24.9	600	−1.2
	Mean	27.5±1.3	950±660	−0.05±0.5
No sweating observed	Co.	27.3	0	+0.6
	Sm.	27.3	900	+0.7
	Ol.	26.2	250	−0.3
	Ro.	26.0	500	+1.0
	Fr.	25.6	500	+0.5−0.3
	Ca.	25.8	0	+0.5
	Mean	26.4±0.7	360±310	+0.4±0.4

minute (5 obs.); in the operating rooms ventilated by open windows, 17 ± 25 f/m (20 obs.); in one room containing 2 fans, 164 ± 15 f/m (4 obs.). In no case could variation in rectal temperature be attributed to the effect of air movement as measured, i.e., at a distance of 6 feet from the patient.

In 12 patients receiving cold blood into a leg vein, rectal temperatures were observed to rise, fall, or remain constant during the time of infusion.

The temperature of the air adjacent to the body under the drapes exceeded the temperature of the air in the cooled neurosurgical theatre by 3.6 ± 1.8° C. (5 obs.), and in the other operating rooms by 3.9 ± 1.0° C. (14 obs.).

Discussion

At any level of activity in the conscious patient or during anaesthesia, the temperature of the interior of the body can be maintained at a constant value only when heat loss to the environment equals heat production or gain. If such a balance does not exist the temperature will change.

Although quantitative information is not available, the existing evidence suggests that heat production in anaesthetized man is less than the basal metabolic rate. Admittedly the quantity of heat produced by skeletal muscle in the basal state is not large (23, 24) but when anaesthesia supplemented by curarization supervenes, muscular relaxation is more complete and the contribution of skeletal muscle to the total heat production is further reduced. The specific dynamic action of foods does not augment heat production because patients entering the operating room have been fasting for at least 10 hours. In addition, further depression of the metabolic rate may be caused by morphine (25, 26) and by certain barbituric acid hypnotics (27). Atropine in doses of gr. 1/50 has been shown to elevate the metabolic rate (28, 29) but smaller doses were used in this study.

When the air temperature falls sufficiently, usually below 19° C. (30, 31), conscious resting man supplements heat production first by increased muscular tension and then by shivering. If muscular relaxation is complete these mechanisms are not available to the anaesthetized patient.

During surgical procedures heat may be gained from the environment from the operating room lamps, from hot laparotomy pads and from the anaesthetic machine when the soda-lime absorber is in use.

Heat loss to the environment occurs by transfer from the skin and lungs, by radiation to distant cooler objects, conduction to the air, and evaporation of water.

Heat exchange by radiation from the skin is a function of the effective radiating surface of the body and the difference between skin temperature and the surface temperature of distant objects "seen" by the effective surface. During surgical anaesthesia radiative loss is therefore assisted by the high skin temperature produced by maximal cutaneous vasodilatation, but hindered by the presence of the hotter operating lamps and by the intervention of the surgeons' bodies between the patient and cooler objects. Transfer by radiation becomes impossible if the temperature of the environment rises above the upper limit of skin temperature.

Thermal conduction to the adjacent air depends upon the area of skin in con-

tact with the air and the surface-to-air thermal gradient. Air movement by natural or forced convection maintains the thermal gradient by replacing the warmed air at the skin surface with cooler air. However, surgical draping creates a "private climate" about the patient's body which is so unsusceptible to erosion by the room air that when air velocities of 50 feet per minute are recorded, the air temperature at a distance of 6-8 cm. from the skin is almost 4° C. above the temperature of the moving air. The result is that while a reclining nude subject can lose heat to the air at temperatures reaching 34° C. (20), conductive heat loss in the operating room becomes seriously impaired as the air temperature approaches 30° C.

Evaporation of insensible perspiration from the skin dissipates about 15 per cent of the total heat loss at air temperatures between 18-30° C., the remainder being lost by radiation and conduction (32). If transfer by these means becomes inadequate, the sweat glands become active. For nude reclining man this occurs at an air temperature of 31° C., when he is lightly clothed at 29° C. (32). For recumbent anaesthetized man under drapes the temperature at which sweating commences is lower, the mean value obtained in this study being 27.5° C.

Sweating is effective as a cooling mechanism because water when evaporated absorbs heat. The rate of evaporation varies inversely with the relative humidity: consequently as the wet bulb temperature approaches the dry bulb, the effectiveness of sweating as a cooling mechanism is reduced. The body can increase the cooling effect by increasing the area of skin that is wet with sweat, and air movement increases the rate of cooling by speeding evaporation. Atropine and scopolamine reduce sweating by as much as 35 per cent (33) and hence limit the capacity of the sweat glands to cool the body.

In each of 10 cases of heat retention during anaesthesia observed by Clark *et al.* (14) cessation of sweating occurred. Searles and Lenahan (12) reversed a hyperthermic trend in an anaesthetized patient by intravenous fluid therapy and Bourne (34) has shown that in dogs etherization at 33° C. produces negligible changes in body temperature when fluids are administered. In this study, although the difference between the mean volume of fluid received by patients who did not sweat was significantly less than the mean volume of fluid received by sweating patients, there was also a significant difference between the mean ambient air temperature for sweating and non-sweating patients, and so perhaps the evidence is inconclusive for establishing a relation between sweating ability and intravenous therapy during surgical anaesthesia. Unfortunately Clark *et al.* (14) have not indicated whether their patients received intravenous fluid.

The lungs dissipate heat chiefly by warming inspired air to 32-35° C. and saturating it with water to 95 per cent (35). For a man with a basal metabolic rate of 71 kg. cal./hour, Clark *et al.* (14) have calculated that by breathing into an anaesthetic machine he loses 16 per cent of his total heat production when the non-rebreathing system is used, 4 per cent when the closed circle system is used, and that he gains 4 per cent when the closed to-and-fro system is employed. A loss of 10 per cent can probably be assumed for the semi-closed partial rebreathing system. Normally vaporization from the skin can increase to compensate for diminished loss from the lungs (36), but when the sweating mechan-

ism is impaired these figures become important. Thus in cases where sweating had ceased and heat retention occurred, the to-and-fro system was most commonly in use (14).

The administration of blood at refrigerator temperature has little effect upon rectal temperature (37).

The exposure of deep tissues through a large skin incision may be an important avenue of heat escape, especially when the air is cool.

In addition, the thermoregulatory centres of the central nervous system may be depressed by the anaesthetic and so become less efficient in their action; certainly the control is seriously disturbed in the anaesthetized child (7).

From these considerations it is not unreasonable to expect that in cool environments the cutaneous vasodilatation and the muscular relaxation achieved during anaesthesia may embarrass the normal response to cold, while in hot environments the described hindrances to radiative, convective, and evaporative heat loss should predispose to hyperthermia, and certainly these situations have developed (20, 38). However, in this study, at air temperatures of 20.1-29.0° C. dry bulb, and 14.8-25.6° C. wet bulb, under the described condition of surgical anaesthesia, only slight variations in rectal temperatures were observed and certainly no serious impairment of thermoregulation occurred.

At an air temperature below 20° C. one case in this study showed a marked hypothermia, but further investigation is necessary to clarify the circumstances under which such changes occur.

Acknowledgment

Dr. S. M. Campbell is thanked for his kindness in sponsoring this study, and for his encouragement and assistance at all times.

Summary

1. Physiological reactions of 35 afebrile adults to the thermal environment during surgical anaesthesia were studied.

2. In the air temperature range 20.1-29.0° C. dry bulb and 14.8-25.6° C. wet bulb observations on the rectal temperature indicated no embarrassment of thermoregulation.

3. The mean air temperature at which sweating occurred was 27.5° C.

4. Skin temperatures along the lower limb rose quickly after induction of anaesthesia to within a few degrees of rectal temperature and generally remained high throughout anaesthesia.

5. Possible factors influencing heat regulation during surgical anaesthesia are discussed.

Résumé

Des enquêtes antérieures sur la température des malades anesthésiés ont suggéré que des troubles de la régulation thermique peuvent se produire et que les fluctuations observées dans la température rectale peuvent être influencées par l'état thermique de la salle d'opération.

Dans la présente étude, on a observé les températures rectales, musculaires et

cutanées et l'activité des glandes sudoripares chez 35 adultes soumis à une anesthésie générale. En même temps on a noté les descriptions des températures prépondérantes (ampoule sèche et ampoule humide) et des mouvements de l'air.

On a constaté que, pour des températures s'étendant de 20, 1° à 29° C. (ampoule sèche) et de 14, 8° à 25, 0° C. (ampoule humide), les températures rectales subissaient peu de variations pendant une intervention durant en moyenne trois heures et que la régulation thermique n'était pas affectée. Les températures cutanées le long du membre inférieur montaient rapidement après le début de l'anesthésie jusqu'à atteindre, à quelques degrés près, la température rectale et en général se maintenaient élevées jusqu'au bout. La température moyenne de l'air lorsque la sudation apparaissait était de 27, 5° C., ce qui est quelque peu plus bas que le seuil de l'homme conscient.

On a discuté les facteurs qui peuvent influencer la régulation thermique pendant l'anesthésie chirurgicale; les principaux étaient la réduction du métabolisme basal et la prévention des frissons obtenue en provoquant la relaxation musculaire complète, la création d'un "climat particulier" autour du corps du malade au moyen de champs opératoires, la relation directe qui peut exister entre la capacité de sudation et l'administration de liquides intra-veineux, et la modification des pertes calorifiques pulmonaires par l'emploi de différentes méthodes respiratoires.

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MANAGEMENT OF CARDIAC ARRHYTHMIAS DURING ANAESTHESIA*

E. L. McCawley, M.D., PH.D.**

When a continuous electrocardiogram is recorded during surgical operations which involve general anaesthesia, 70 to 80 per cent of the patients will show disorders of cardiac rate and rhythm. Most of these arrhythmias have little clinical importance. To be feared are those which lead to a decline in blood pressure or those which may be a prelude to ventricular fibrillation or cardiac arrest. Specific treatment with antiarrhythmia drugs requires first an accurate diagnosis, reached by interpretation of the electrocardiogram. Observation of the pulse by palpation or auscultation during blood pressure measurements provides little information on the presence or nature of cardiac arrhythmias.

There are two situations which focus the attention of the anaesthetist on cardiac arrhythmias. First, "poor-risk" patients are not denied needed surgery because they have cardiovascular disease. Then, more recently, the manoeuvres devised for repair of cardiac valves or septa are accompanied by ominous changes in cardiac rhythm. The anaesthetist is accustomed to coping with rapid changes in the patient's physiological status, and is in the best position to provide therapy when there is deterioration of cardiac function. While the cardiologist should be consulted freely, he rarely can be present throughout surgical operations.

Anaesthetic Agent and Disturbances of Cardiac Rhythm

During ether or nitrous oxide anaesthesia cardiac arrhythmias are usually of the innocuous type arising chiefly from the supraventricular centres. Sino-atrial or atrioventricular nodal tachycardias are the most common. Atrial extra systoles may also appear. Serious arrhythmias, whose origins are in the ventricles, are seldom evident. During induction of anaesthesia with chloroform, slowing of cardiac rate (bradycardia) and cardiac arrest may occur. With the thiobarbiturates also, Pentothal, Surital or Kemithal, there are episodes of sinus arrhythmia and slowing of cardiac rate. These arrhythmias, seen with chloroform, trichloroethylene, or the thiobarbiturates, may be provoked in experimental animals by stimulation of the vagus nerve. It has been assumed that, until an adequate depth of anaesthesia has been reached, vago-vagal and tracheocardiac reflexes are elicited on passing an endotracheal tube or caused by the irritating nature of the anaesthetic gas mixture. During deep chloroform anaesthesia the vagus is quiescent and aberrant ventricular systoles may appear at that time. However, the ventricular fibrillation, which occurs with animals who are given adrenalin during chloroform administration (1), is seldom observed in man. As more experience has been gained with cyclopropane, there has been a greater facility in the control of the ventricular arrhythmias which accompany its use. Nevertheless, ventricular tachycardia (and possibly ventricular fibrillation) can occur with

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any duration or depth of anaesthesia with cyclopropane if adrenalin is released or administered. In experimental animals a dose of 10 microgm./kgm. of adrenalin suffices to cause ventricular arrhythmias—it is believed that this amount can be released from a patient's adrenals by manipulations in that area. Thus, chloroform or cyclopropane anaesthesia is hazardous during operations for pheochromocytoma.

Hypoxia and Hypercarbia as Factors Initiating Cardiac Arrhythmias

Denson and Joseph (2) attribute the majority of the arrhythmias which occur during induction to hypoxia. The arrhythmias consisted of premature ventricular systoles and ventricular tachycardia. All arrhythmias reverted to normal sinus rhythm on improvement of pulmonary ventilation. The duration of apnea which resulted from endotracheal intubation was only 18 to 71 seconds. They suggest that tracheal-initiated reflexes are not involved because the appearance of the arrhythmias was delayed for several minutes after passing the tube. Johnstone (3) also reports that hypoxia provoked arrhythmias in all patients and in varying stages of anaesthesia. Again, the predominant arrhythmias seen were coupled ventricular extrasystoles and ventricular tachycardia. He stresses the view that spontaneous respirations during cyclopropane should be assisted at all times. Johnstone also reports that the accumulation of carbon dioxide (hypercarbia) is accompanied by ventricular arrhythmias in most patients. Removal of the soda-lime absorber for periods of time (as short as 10 to 15 minutes) will stimulate changes in cardiac rhythm. Hypoxia and accumulation of carbon dioxide are so frequently the cause of cardiac arrhythmias that some anaesthetists teach that no antiarrhythmia drugs are needed, provided due attention is paid to maintaining an adequate oxygen-carbon dioxide balance.

Classification of Disturbances of Cardiac Rhythm

Since identification of the cardiac arrhythmia must precede rational therapy, a review of the classification of arrhythmias is pertinent. Arrhythmias may arise from the sino-atrial or atrioventricular nodes or be atrial or ventricular in origin. Activity of the vagus will inhibit the sino-atrial node; bradycardia, sinus pauses, and even complete asystole may result. If preanaesthetic medication with belladonna alkaloids is inadequate or the patient's vagal reflex unduly sensitive, the intravenous administration of a Banthine-like drug is suggested. Vagal-induced asystole apparently does not occur without cardiac ischemia or excessively deep anaesthesia. The prophylaxis and management of this cardiac emergency has been admirably discussed in a recent issue of this journal by Fortin and Letienne (4). The term supraventricular tachycardia focuses attention on a rapid cardiac rate. It includes rapid rhythm and arrhythmias arising from either node or the atrial myocardium (atrial flutter or fibrillation). Supraventricular arrhythmias generally require no treatment unless the patient's cardiac reserve is minimal. If a supraventricular tachycardia is allowed to persist in these patients, evidence of a "strain" pattern may appear in the electrocardiogram. As a result of cardiac ischemia a coronary infarct may be precipitated. Then, too, the rapidly beating heart may exceed its compensation and acute congestive failure can be the result. A rapidly acting digitalis glycoside may be employed, the object being

to ensure a slow, albeit still irregular, rhythm. Although cardiac output and efficiency usually follow successful conversion to normal sinus rhythm from atrial fibrillation or flutter, quinidine therapy during anaesthesia is too dangerous. In fact, because of the high oxygen tensions possible during anaesthesia, auricular fibrillation may disappear. Aberrant ventricular systoles, even if coupled into bigeminal or trigeminal rhythms, are treated only because it is believed that they are premonitory of ventricular tachycardia or ventricular fibrillation. During ventricular tachycardia blood pressure falls and with ventricular fibrillation there is circulatory failure. Procaine amide is the drug of current choice to arrest ventricular extrasystoles as prophylaxis against the serious ventricular arrhythmias. Ventricular fibrillation requires opening of the thorax, cardiac massage, and use of electrical defibrillators.

Pharmacology of Antiarrhythmia Drugs

Procaine has for some years been applied topically by the surgeon prior to manipulations of the heart and great vessels, with a reduction of cardiac arrhythmias. When it was realized that procaine could be given intravenously, this route of administration was adopted for prophylaxis of cardiac arrhythmias. Since procaine is rapidly hydrolysed and inactivated by serum esterases, it must be given by continuous drip to be effective. Procaine amide (Pronestyl) was synthesized as a substitute for procaine as it is slowly destroyed in the body—its duration of action may be as long as 3 hours. Diphenhydramine (Benadryl) is chemically quite similar to procaine and has a very stable ether linkage. Benadryl has proven effective in preventing ventricular tachycardia or ventricular fibrillation in experimental animals (5) and clinical trial is indicated. Pronestyl is primarily of value in the control of ventricular arrhythmias. Peters, Newman, and Burstein (6) employed a dose of 300 mgm. of Pronestyl and found a significant reduction in the number of arrhythmias which occurred during induction of anaesthesia with cyclopropane. Joseph and coworkers (7) recommend its use for prophylaxis and therapy of ventricular arrhythmias during thoracic surgical procedures. There is, however, doubt that Pronestyl can actually prevent ventricular "flutter" or ventricular fibrillation. If it cannot, then there is little justification for its use in control of alarming but not serious premature ventricular systoles. Experimentally induced ventricular fibrillation in animals may be controlled only by doses of Pronestyl in excess of 40 mg./kg. (a dosage in excess of that permitted in patients). Pronestyl is to be given intravenously no more rapidly than 200 mgm. (2 cc.) per minute and no more than 0.5–1.0 gm. at any one time. Even with this dose hypotension may occur, although the drop in systolic pressure does not usually exceed 40 mm. of Hg. An additional side action of Pronestyl is the result of slowing of myocardial conduction. Following an overdose, bundle branch block may appear and proceed to ventricular fibrillation. If the QRS interval is prolonged more than 20 per cent of the initial value, drug injection should be stopped. Pronestyl also may prolong the QT interval and flatten, notch, or invert the T wave. Despite its defects, Pronestyl is currently preferred to quinidine gluconate. Hypotension and conduction disturbances with intravenously administered quinidine are common.

Rapidly acting digitalis glucoside preparations are frequently needed to slow

the heart rate with supraventricular tachycardias. Usually lanatocide C (Cedelanide) or ouabain are employed. With these glycosides the onset of action may require 10 to 30 minutes, the peak effect being delayed for 45 to 180 minutes. With drugs having such a long lag period before full action develops, overdosage or underdosage will be a common occurrence. Recently, acetyl strophanthidin has been introduced. This drug has a rapid but brief action; the new digitalis substance, when injected intravenously, has an onset of action in ¼ to 5 minutes and peak effects within 10 to 12 minutes. Potentially dangerous effects from an overdose persist but 30 minutes. Currently (8) this new drug is being employed as a "tolerance test" to ascertain the degree of digitalization of patients whose digitalis status is in doubt. Enselberg and coworkers (9) have found it valuable to control the rapid arrhythmias arising during surgical operations. The myocardial sensitivity to digitalis may change abruptly as a result of stresses, fluid and ionic changes which occur during surgery. Acetyl strophanthidin should prove a valuable aid to the anaesthetist in the control of rapid rhythms or acute congestive failure which may develop.

Digitalis overdosage results in a variety of cardiac arrhythmias, the most common being ventricular extrasystoles and ventricular tachycardia. It is conceivable that during surgery the patient's myocardial sensitivity to digitalis may increase and account for these and other arrhythmias. Toxic doses of digitalis liberate potassium from within the myocardial cell. Loss of potassium will occur through the kidney or through fluid balance shifts. Administration of potassium promptly reverses the ventricular arrhythmias of digitalis overdosage (10). Potassium chloride may be injected slowly intravenously until the arrhythmia ceases or evidence of hyperkalemia appears in the electrocardiogram. Toxic doses of potassium, hyperkalemia, produce a characteristic high, peaked T-wave and beginning signs of heart block. If administration of potassium be continued, complete electrical depolarization of the heart will take place. This toxic action has been adapted for therapeutic purpose. Ventricular fibrillation may occur during hypothermic anaesthesia. Swan and Virtue inject potassium into the fibrillating heart until complete depolarization occurs—the excess potassium is flushed into the circulation by direct masage of the heart. If normal spontaneous rhythm does not appear as a result of the massage the injection of calcium gluconate usually produces the desired effect. In their experience the use of electrical defibrillators has been disappointing. The role of potassium on the actions of the heart is still poorly understood but should be considered in evaluating clinical cardiac arrhythmias.

Summary

Disorders of cardiac rhythm may be described as supraventricular or ventricular in origin. The former for the most part are innocuous. Ventricular premature systoles may be the forerunner of ventricular tachycardia or ventricular fibrillation. Ventricular tachycardia if persistent is accompanied by failing circulation. The majority of the arrhythmias observed during anaesthesia can be attributed to hypoxia or accumulation of carbon dioxide. In the opinion of certain anaesthetists, if these two causes of arrhythmia are avoided there is no need for an antiarrhythmia drug. Procaine amide has proven of value in the control of the

ventricular arrhythmias which occur during cyclopropane anaesthesia. Rapidly acting digitalis preparations, particularly acetyl strophanthidin, may be needed to slow a rapidly beating heart (supraventricular tachycardias). The ventricular arrhythmias which accompany digitalis toxicity are caused by loss of intracellular potassium. Potassium chloride is effective in the control of digitalis-induced arrhythmias and should be considered in other instances as well.

Résumé

On peut décrire les troubles du rythme cardiaque comme étant d'origine supra-ventriculaire ou ventriculaire. Les premiers sont pour la plupart inoffensifs. Des systoles ventriculaires prématurées peuvent être les signes avant-coureurs d'une tachycardie ou d'une fibrillation ventriculaires. Si la tachycardie ventriculaire persiste, elle s'accompagne d'insuffisance cardiaque. La majorité des arythmies observée en cours d'anesthésie peuvent être attribuées à l'hypoxie ou à l'accumulation d'anhydride carbonique. Selon certains anesthésistes, si l'on évite ces deux causes d'arythmie, il n'est nul besoin d'employer une médication contre l'arythmie. La procaine amide a été reconnue utile pour juguler les arythmies ventriculaires qui se produisent pendant l'anesthésie au cyclopropane. On peut avoir besoin de préparations à action rapide à base de digitale, en particulier de l'acétyl-strophantidine, pour ralentir les battements du cœur trop rapides (tachycardie supraventriculaire). Les arythmies ventriculaires qui accompagnent la toxicité de la digitale sont dues à une perte de potassium intra-cellulaire. Le chlorure de potassium est efficace pour juguler les arythmies provoquées par la digitale et son emploi doit être aussi bien envisagé dans d'autres cas.

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RESPIRATORY PHYSIOLOGY IN RELATION TO ANAESTHESIA*

FERNAND GRÉGOIRE, M.D., F.C.C.P.**

IT is common knowledge that the purpose of respiration is to furnish to the tissues the oxygen needed and to remove the carbon dioxide produced by the metabolism. It seems evident that every anaesthetist tries to respect these purposes and to fulfil these requirements. Notwithstanding the progress made in recent years, the occasional patient succumbs near the end of operation or shortly thereafter, or more often barely survives a puzzling condition of low blood pressure. At such times it is customary to say: "The blood replacement was not sufficient," or "his heart just would not stand it," and so on.

Stand what is often not clear. We shall try here to review briefly some general data on respiratory physiology, in normal and in emphysematous patients, and to describe an acidosis of potentially fatal degree which results from accepted procedures in thoracic and general surgery. We shall then try to find a way to correct it and to prevent the complications which follow it.

The normal respiration is under the control of the respiratory centre which may be influenced directly or reflexly. The neural mechanisms responsible for rhythmic respiration can be divided into basic and accessory parts.

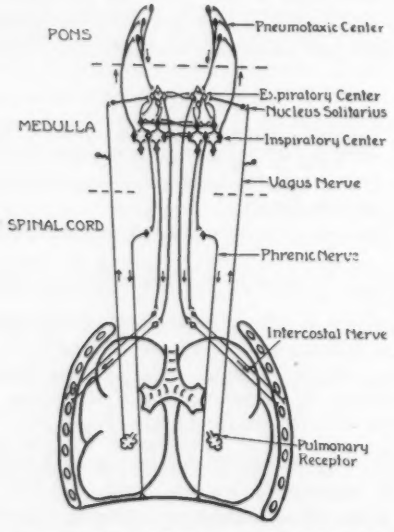

Fig. 1. Diagrammatic representation of basic neural mechanisms responsible for rhythmic respiration.

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Basic Neural Mechanism (Fig. 1)

A. Medullary respiratory centre
   Inspiratory
   Expiratory
B. Pneumotaxic centre in the lower pons
C. The Hering Breuer reflex or distention receptors in the lungs

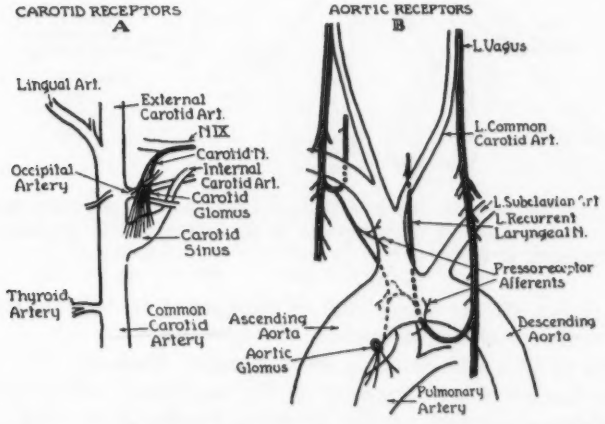

Fig. 2. Diagrammatic representation of location and neural connections of carotid and aortic chemoreceptors and pressoreceptors (dog). From Comroe, Am. J. Physiol. *127*: 176 (1939); and Comroe and Schmidt, Am. J. Physiol. *121*: 75 (1938).

Accessory Neural Mechanism (Fig. 2)

Carotid and Aortic chemoreceptors
Carotid and Aortic pressoreceptors
Protective reflexes
Voluntary control

Fig. 3 shows the control of the respiration by the respiratory centre, which is influenced chemically and reflexly.

An increase of CO_2 stimulates the respiration. If a normal subject is given 3 per cent CO_2 to breathe, his ventilation may double, and the pCO_2, normally 40 mm Hg., may increase by only 2.5 to 3 mm. Without this hyperventilation, the pCO_2 would rise to 60 mm Hg. and there would be an important acidosis.

When 9 per cent CO_2 is inhaled by a normal subject, the ventilation is increased about ten-fold, and the pCO_2 will go as high as 65 or 68 mm Hg. with a pH around 7.00.

If more than 9 per cent CO_2 is inhaled, there will be some depressant effect on the respiratory centre and at higher concentrations, i.e., above 70 mm Hg. in the alveolar air, the CO_2 becomes anaesthetic and may even produce lethal effects. The barbiturates, opiates, and anaesthetics are also depressors of the

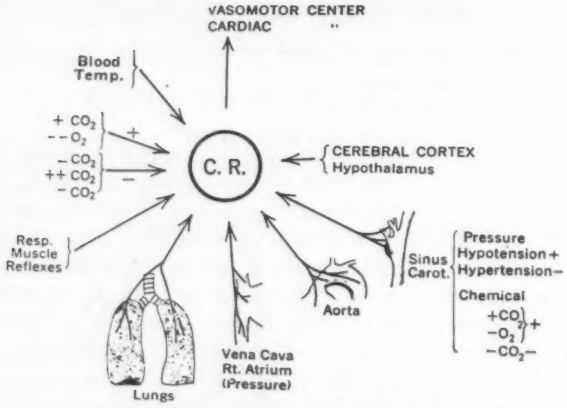

Fig. 3. Diagram indicating the influence of O_2 and CO_2 in direct and reflex modifications of the respiratory centre according to conceptions of D. Cordier and C. Heymans (Le Centre respiratoire, Herrmann et Cie).

respiratory centre. On the other hand an increase of O_2 will not influence the respiratory centre of *normal* subjects.

A decrease of CO_2 will diminish the respiration. Similarly although surprisingly, a decrease of pO_2 is depressor on the respiratory centre and it is only through the chemoreceptors, i.e. the carotid and aortic glomi, that anoxia stimulates the breathing. The pO_2 must drop by at least 20 mm Hg. to produce a stimulation of the chemoreceptors. An increase of pCO_2 and a decrease of pH have also an action on the chemoreceptors, but much less than on the respiratory centre.

The pressoreceptors also play a role, but are more efficient on circulation than on respiration. We know, however, that a sudden rise in blood pressure causes apnoea and that a sharp fall produces hyperpnoea.

The Hering Breuer reflexes are very important in the reflex control of breathing. These nerves are distributed to bronchioli, alveolar ducts, and alveoli. They are essentially distention reflexes. During inspiration, they send impulses to the respiratory centre and when enough impulses are accumulated there is inhibition of the continuous discharge of the inspiratory centre and expiration starts. The greater the resistance in the lungs, due to fibrosis, congestion, or cardiac oedema, the more numerous are the impulses and the faster is the breathing.

In Fig. 4 we have an idea of what happens in a normal subject with an R.Q. of .83. The pO_2 is around 100 mm Hg. and the pCO_2 40 mm Hg. The pCO_2 closely follows the ventilation. By hyperventilation there is pCO_2 fall and pO_2 increase with a high R.Q., and a high pH going towards alkalosis.

On the other hand, hypoventilation prevents the renewing of the air in the alveoli, and the CO_2 keeps increasing, poured in from the venous blood. The pCO_2 in the alveoli and in the blood gets into equilibrium, and as the pCO_2 increases the pH of the blood becomes more and more acid.

Fig. 5 illustrates the nitrogen rinsing curves of three subjects. The subject breathes pure O_2 for 7 minutes in an opened circuit, so that at the end the per-

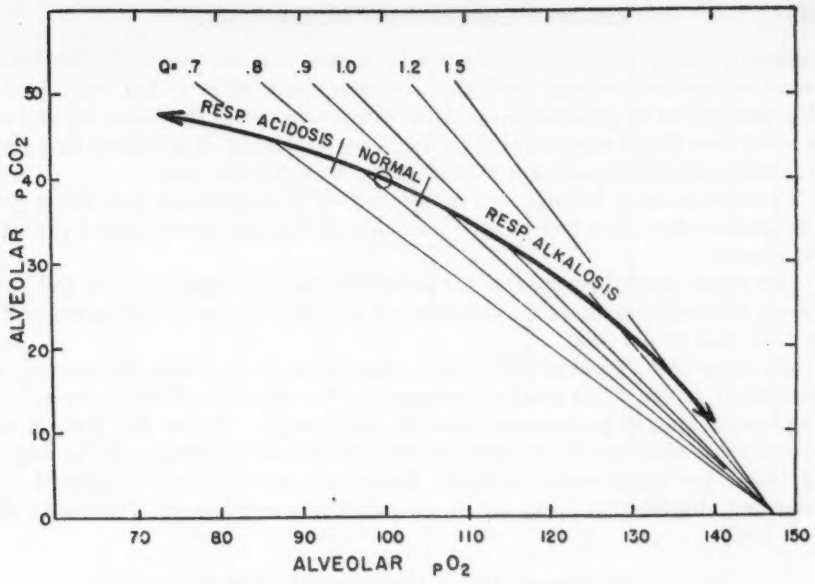

Fig. 4. Respiration in a normal subject.

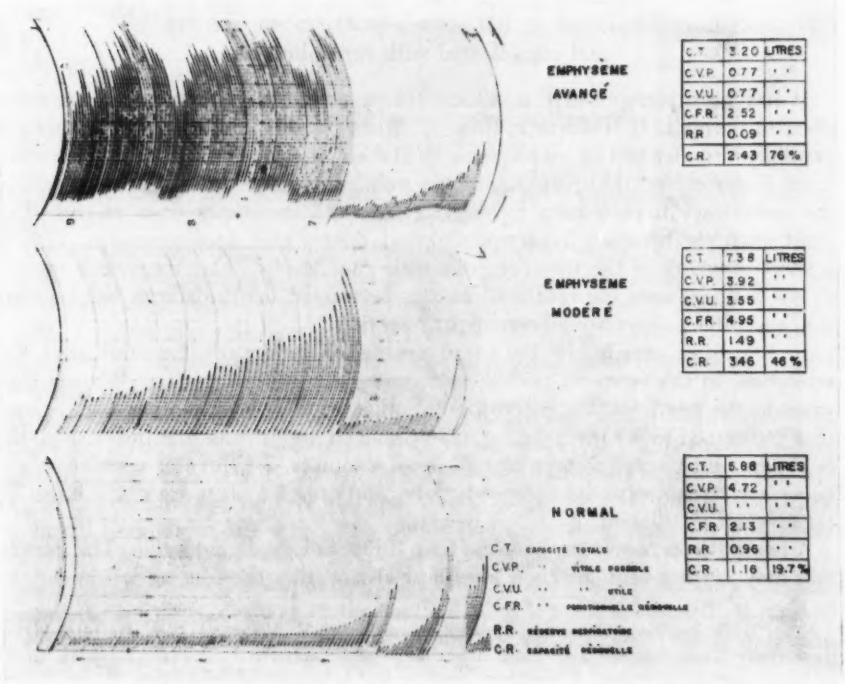

Fig. 5. Nitrogen rinsing curves of normal and emphysematous subjects.

centage of N_2 in his lungs should not be higher than 2.5 per cent. The lower curve represents a normal individual. Starting from right to left, we see the first part on the 80 per cent scale. After 1½ minutes, when the N_2 in expired air is lower than 10 per cent, we shift to the 10 per cent scale. It is evident that after 3 minutes the concentration is already lower than 2.5 per cent.

The second curve belongs to a moderate case of emphysema and shows that this patient takes more time to rinse his lungs. At the end, there is still 4 per cent N_2 present.

The upper curve belongs to a far advanced case of emphysema. At the end, during a forced expiration for collection of alveolar air, the concentration of N_2 is more than 10 per cent.

We know that a residual air of more than 40 per cent (when this increase in percentage is due to an absolute increase) is the signature of emphysema. The emphysema is more pronounced when the percentage is higher. But there is not always a complete correlation between the residual air in percent and the degree of blood gas impairment. Baldwin, Cournand, and Richards suggested the following classification in an article on pulmonary insufficiency (Medicine 28: 210, 1949).

1st = Saturation at 92% or above during moderate exercise.
2nd = Saturation $<$ 92% with a $pCO_2 <$ 48 mm Hg.
3rd = Saturation $<$ 92% with a $pCO_2 >$ 48 mm Hg.
4th = Saturation $<$ 92% with a $pCO_2 >$ 48 mm Hg.
and complicated with cor pulmonale.

It has been recognized that anoxia increases the pulmonary artery pressure even in normals. It is worse if there is already a diminution of the pulmonary capillary vascular bed by emphysema or fibrosis. If a lobectomy or a pneumonectomy is performed the reduction of the pulmonary capillary bed is greater and the pulmonary hypertension is likely to develop, sometimes even at rest. The right ventricle becomes hypertrophied and dilates and decompensates. This is a short summary of the way cor pulmonale can develop after thoracic surgery.

We shall see now the results of studies performed on 19 patients before, during, and after pulmonary lobectomy or resection.*

In Fig. 6 we can follow the mean ventilation before, during, and after the operation, in the recovery period, and during the following days. We see that prior to the operation the mean total ventilation was 6.8 litres per minute and that it dropped to 4.8 litres during anaesthesia. If we assume that the dead space is 150 cc. and the respiratory rate 16 times a minute, 2.4 litres of ventilation are necessary just to wash out the dead space, and only 2.4 litres are really effective for alveolar ventilation.

A few patients, however, had less than 3 litres of total ventilation. This means that they did not ventilate their alveoli at all and that they succeeded in surviv-

*This work has been done in collaboration with Dr. E. Bousquet and Dr. L. Longtin respectively Chief Surgeon and Chief Anaesthetist at St. Joseph Sanatorium Hospital, aided by federal-provincial grants. A complete report is now in process of publication.

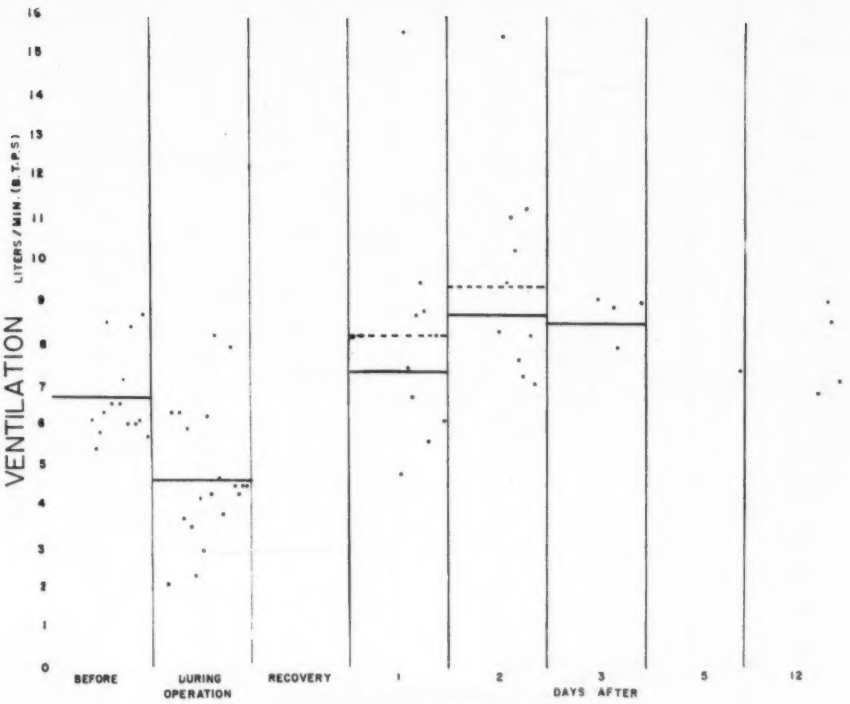

Fig. 6. Total ventilation and pulmonary resection.

ing with diffusion respiration. As the CO_2 follows the ventilation closely we can suspect what influence this hypoventilation will have.

During the following days the ventilation is slightly higher than before. It is important to note that none of these patients was emphysematous and that they all received ether anaesthesia with oxygen.

Fig. 7 shows that the saturation as a mean was higher during the operation than after or before. It is evident that the first purpose of respiration, i.e. the provision of O_2 to the tissues, is fulfilled.

Fig. 8 shows the CO_2 content of the arterial blood. As a mean, before operation it was of 47 Vol. per cent with extremes between 42 and 50 Vol. per cent. During operation, the mean was 60.50 Vol. with extremes between 52 and 72 Vol. per cent. It is evident that a great accumulation of CO_2 occurred during operation. It started to diminish in the recovery period and maintained itself slightly higher than normal in the following days.

In Fig. 9 the effect of hypoventilation is still more evident on the pCO_2 determined directly, according to the method of Riley and Prommell. It was 39 mm Hg. as a mean before, with 32 to 45 mm Hg. as extremes. During operation the mean pCO_2 was 90 mm Hg. with extremes from 62 to 126 mm Hg. It went

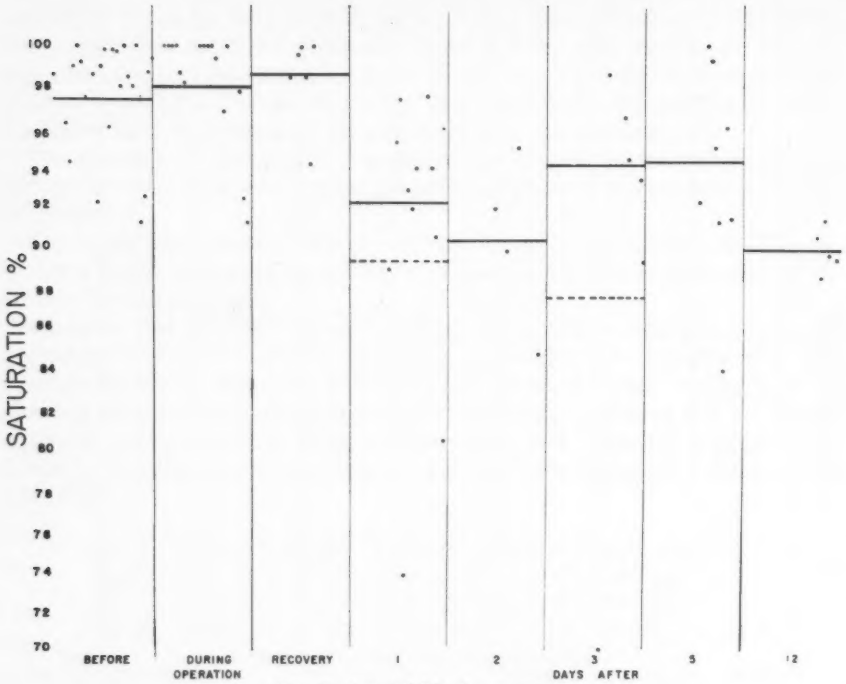

Fig. 7. Arterial blood saturation.

back to previous levels the following day, but this decrease started in the recovery period, one hour after the end of anaesthesia, when the patient had already begun to hyperventilate.

With an increase in pCO_2 and in CO_2 content there is respiratory acidosis (Fig. 10). PH determinations were made on a Cambridge pH meter and a fall in pH was found. It passed from 7.42 before, to 7.14 with one at 6.95 and another one at 7.00 during operation. It was found at 7.26 one hour after operation and back to previous levels two days after.

In Fig. 11 we have a summary of the effect of hypoventilation on acid base balance equilibrium during anaesthesia.

Many investigators have found the same kind of acidosis (1) (2) (3) (4) (5) (6) (7). But what are the effects of this acidosis? Goldstein and DuBois (8) have shown that a *fall of blood pressure* immediately follows the cessation of breathing high concentrations of CO_2.

Dripps (9) has discussed the fall of blood pressure which occurs when the subject is taken off cyclopropane. He reported that under this agent the pCO_2 rises to great heights; a considerable fall of blood pressure sometimes occurs when the cyclopropane is terminated. "The higher the tensions of the CO_2 in the arterial blood, the greater is the likelihood of a postanesthetic decrease in blood pressure."

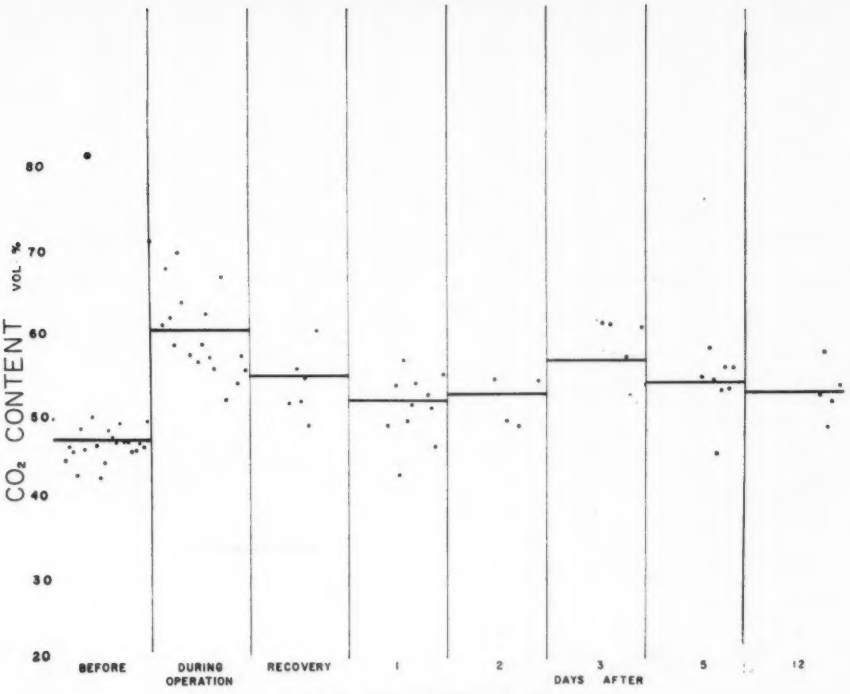

Fig. 8. CO_2 content of arterial blood.

Beecher and Murphy (1) said: "When the pH rises and the pCO_2 falls, its stimulant action is withdrawn and the blood pressure falls. As pointed out previously, there is reason to believe that this is a true cause and effect relationship."

Dr. Sealy and collaborators (6) in their paper entitled "Studies on Cardiac Arrest: The Relationship of Hypercapnea to Ventricular Fibrillation," state: "*Sudden cardiac arrest* constitutes one of the chief causes of death during surgery. If hypoxia, overdosage of drugs or peripheral vasometer collapse may be the known precipitating factors in some instances, the majority of cases have to be classified as due to *undetermined* causes."

Since several observers such as Campbell (10), Mayer and associates (2), Young and associates (11), Miller, Brown *et al.* (3) had already suggested that hypercapnia may be one of the responsible factors in this group, Sealy, Young and Harris (6) performed a group of experiments in their laboratory on dogs exposed to 30 and 40 per cent CO_2. They stimulated the intact right vagus nerve until "vagal escape occurred." It was noted that the duration of the asystole was increased and varied directly with the degree of hypercapnia.

The dog's tolerance to severe respiratory acidosis was notable. In three instances out of nine, however, ventricular fibrillation was noted, when after a

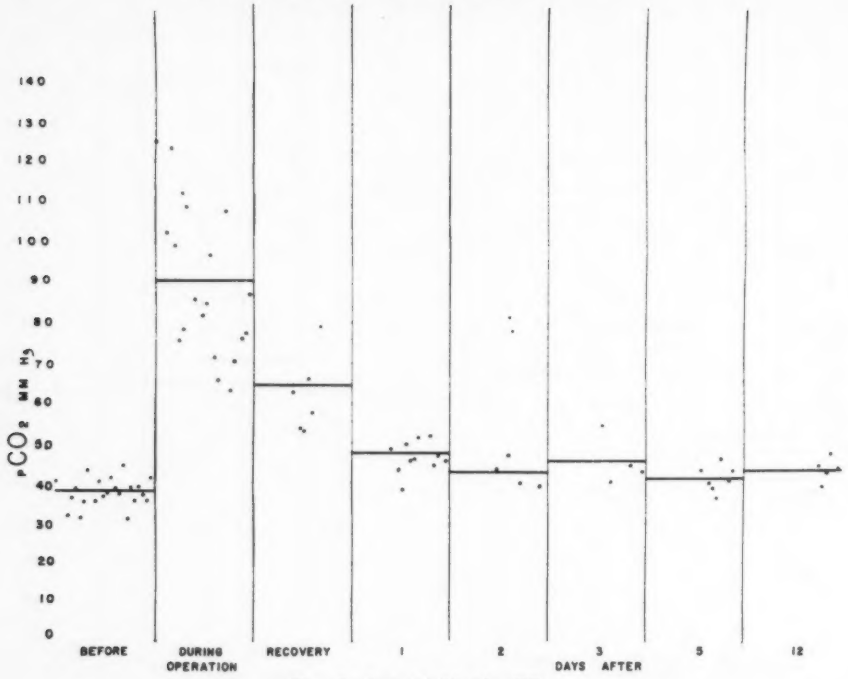

Fig. 9. Arterial blood pCO_2.

long period of hypercapnia the dog was allowed to breathe room air. Significant alterations in the electrocardiogram always occurred before ventricular fibrillation. These included alterations in T waves, increase in the intraventricular conduction time, ventricular extrasystoles and A-V dissociation.

Because of the similarity of the electrocardiographic changes of hyperkalemia with those in the post-hypercapneic period, the same workers performed another group of experiments using rats. The plasma potassium increased in proportion to the length of the hypercapneic period. The records of patients who developed cardiac arrest were then studied. Out of 36 cases, 24 had cardiac arrest from unexplained causes. It was considered possible that hypercapnia could have occurred in ten cases of abdominal surgery and 11 cases of thoracic surgery in the middle portion of the operation.

Brown and Miller (12) in 1952 were the first to show the exact significance of ventricular fibrillation in the immediate post-hypercapneic period in dogs. The rise in potassium levels after cessation of hypercapnia was also noted and may be due to a sudden release of adrenaline.

The hurried administration of large quantities of blood to a patient in whom ventilation and renal excretion are impaired would seem to offer a good opportunity for the occurrence of toxic levels of plasma potassium. The low pH of the transfused blood is possibly an adjuvent to the acidosis.

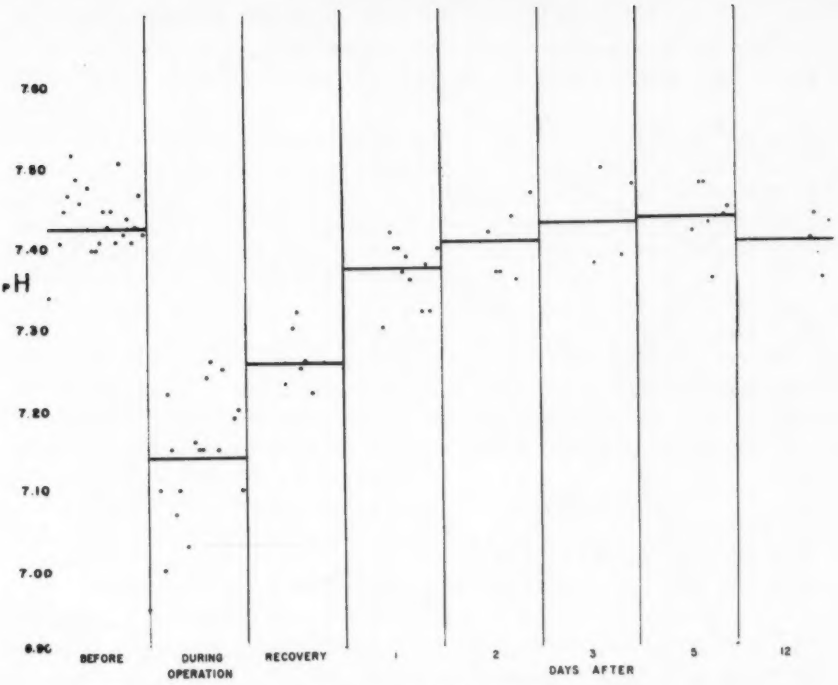

Fig. 10. Arterial blood pH.

There are drugs such as ether which significantly decrease the blood levels of potassium. It is a plausible assumption that this agent may actually protect the heart by reducing the levels of the plasma potassium. The most important measure of prevention of respiratory acidosis is an adequate ventilation during anaesthesia.

Donald Proctor in an article on respiration and anaesthesia in *Surgeon*, September, 1953, stated: "It is remarkable that in this age of exact measurement the anesthetist is generally forced to estimate the adequacy of his patient's ventilation by observing or feeling the excursions of a rubber bag which, when full, holds 5 litres of gas. Even the most skilled and experienced person cannot hope to estimate such excursions accurately. As a result, most of us have relied on our ability to err on the side of overventilation. Even a superficial perusal of recent literature suffices to demonstrate how frequently the error occurs on the other side."

Our results are in full agreement with this. It seems that adequate ventilation during operation and a gradual return to room air after anaesthesia to prevent a rapid fall of CO_2 are absolutely necessary to avoid the complications of acidosis. If at the end of the operation, hyperventilation may happen in a normal subject from the respiratory point of view, on the other hand there is the danger of

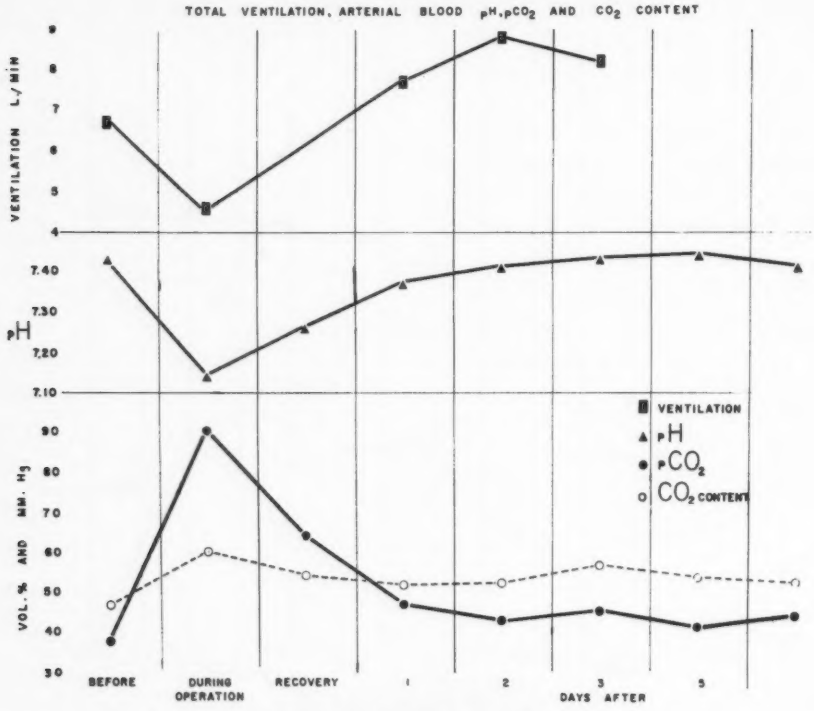

Fig. 11. Total ventilation, arterial blood pH, pCO_2 and CO_2 content.

hypoventilation in the emphysematous patient. The patient who does not succeed in maintaining a normal CO_2 content, a normal pCO_2 and pO_2 before operation, may have much more difficulty after it.

The CO_2 content and the pCO_2 will be increased more, and as soon as the patient starts to breathe room air, he has trouble because of his emphysema. If he has had thoracic surgery he may have paradoxical respiration and very bad ventilation.

These patients cannot get rid of their secretions because of the lack of elasticity and the incapacity to build up enough positive pressure in their lungs. These secretions obstruct the airway and become an impairment to the ventilation. The patient becomes anoxic, the CO_2 increases in his blood, the pulmonary artery pressure increases, and there may be failure of the right heart.

If pure oxygen is given, the patient immediately feels better, his minute ventilation diminishes as a result of the saturation of the arterial blood, and it may happen that the respiration stops because the reflex stimulation of the respiration due to anoxia on the aortic and carotid glomi does not exist any more. More frequently, however, the respiration will continue, but is much diminished; and even if the blood becomes well saturated with pure O_2, there is accumulation of CO_2, respiratory acidosis, coma, and death.

How can we prevent this?

The first thing to do is to have a physiological evaluation of the respiratory status of the subjects who have had chronic pulmonary disease prior to anaesthesia.

If the patient is emphysematous in the third or fourth degree, he should have general anaesthesia only if it is impossible to give him another kind. Then the anesthetist should use a mechanical ventilatory device or control the respiration manually, check the minute ventilation and be sure there is no hypoventilation. A CO_2 content and pH determination are very important and should be done regularly in these patients to prevent increased acidosis.

It is contra-indicated to "aid" the patient's respiration by the continuous inflation of the breathing bag with a slight amount of positive pressure (for example, 3 to 5 cm. of water). Measurements made by Maloney, Derrick, and Whittenberger (13) demonstrated that this *continuous positive pressure* actually decreased the minute ventilation by 40 per cent on the average. This decrease in ventilation was in addition to the decrease from 8.6 litres to 4.4 that had already occurred when the pleura was entered.

There are several reasons for this effect of continuous inflation:

1. It is mechanically more difficult for the normal lung and still more for the emphysematous one to breathe against the resistance of a distended bag.
2. Continuous pressure causes a reflex slowing of the respiratory rate in many patients.
3. The continuous positive pressure decreases the venous return to the heart, increases the filling time of the heart, and diminishes the stroke volume with a fall in blood pressure if the pressure of O_2 is more than 7 cm. of water.

In patients anaesthetized in the usual way the finding of tidal volumes which were inadequate to clear the airway dead space was indeed surprising.

Draper, Whitehead, and Spencer (14) have studied diffusion respiration with animals breathing pure O_2. They found that the oxygenation remained good, but the CO_2 accumulated.

It seems that this technique is frequently, although unintentionally used, especially during thoracic anaesthesia.

One can easily see how dangerous it is to give 3 to 5 per cent CO_2 mixtures to breathe after operation. There is danger of still further increasing the acidosis except in cases which have been hyperventilated.

It seems evident that we know more and more about the possible complications of anaesthesia, but there is still place for research. In conclusion, let me quote Dr. H. J. V. Morton (15): "Since the earliest days in anaesthesia, respiration has provided helpful signs for those who conduct fellow human beings on journeys through unconsciousness. We have no reason to suspect that the last secret has been revealed; that no more useful information is forthcoming. Let us then apply ourselves with renewed vigour to the study of respiration and progress in anaesthesia will surely result."

Summary

A general review of the nervous and chemical controls of the respiration has been made.

We have then presented data on ventilation and arterial blood, before, during, and after pulmonary resections in 19 cases, showing that hypoventilation exists and is responsible for the respiratory acidosis that always accompanies the increase in CO_2 content and in pCO_2.

The effects of CO_2 retention on heart and central nervous system during anaesthesia and in the recovery period have been reviewed.

It was suggested that hypoventilation be prevented by means of a ventilatory mechanical device or a manual control with a flow meter, maintaining the ventilation at the preanaesthetic level.

It was also suggested that physiological tests might be made on subjects who have chronic respiratory disease in order to find their respiratory reserves.

Résumé

Une revue générale des contrôles nerveux et chimiques de la respiration a été faite.

Nous avons ensuite présenté les données de la ventilation et du sang artériel avant, durant et après des résections pulmonaires chez 19 individus et avons montré qu'une hypoventilation marquée existe et est la cause d'acidose respiratoire marquée dans certains cas.

Les effets de rétention de CO_2 avec chute du pH sur le cœur et sur le système nerveux central durant l'anesthésie et dans la période de recouvrance ont été analysés.

On a alors suggéré de prévenir l'hypoventilation au moyen d'un appareil mécanique avec pompe, ou à défaut de cet appareil, de contrôler manuellement la respiration à condition de pouvoir en mesurer le débit.

L'importance d'établir le status physiologique respiratoire des patients qui souffrent de maladies pulmonaires chroniques a aussi été indiqué.

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SUCCINYLCHOLINE–NITROUS OXIDE ANAESTHESIA FOR MITRAL COMMISSUROTOMY* **

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DURING the past decade, cardiac surgery has progressed from the realm of the occasional operation to an established position in the surgical armamentarium. While surgical interventions upon the heart are not commonplace, the performance of these procedures has become sufficiently widespread to necessitate an understanding of the inherent problems involved by all those engaged in the surgical care of these patients. These problems vary considerably in relation to the individual cardiac lesion for which surgical therapy is undertaken: in the instance of surgical attack upon mitral stenosis by commissurotomy (or valvulotomy), they impose unique demands upon not only the surgeon and the cardiologist, but also upon the anaesthesiologist.

An intelligent approach to the anaesthetic management of the patient undergoing mitral commissurotomy must be based upon a detailed knowledge of the pathologic physiology of mitral stenosis. A number of different anaesthetic techniques have been evolved and described (1–5). The anaesthetic agents chosen are of far less importance than a strict adherence to certain fundamental tenets of anaesthetic management which have followed from a rational understanding of the profound physiological aberrations associated with mitral valvular disease. It will be the purpose of this presentation to describe an anaesthetic technique which employs succinylcholine–nitrous oxide anaesthesia to overcome a number of the very real difficulties imposed by the operation of commissurotomy in these seriously ill patients.

THE PATHOLOGIC PHYSIOLOGY OF MITRAL STENOSIS

Mitral stenosis has its origins in the initial attack of acute rheumatic fever, which occurs, typically, during the first or second decade of life. There follows a relatively asymptomatic period of from several to twenty or more years during which the endocarditis produces a progressive fibrosis and contraction of the mitral valve. Mild dyspnoea on exertion occurs, associated with pulmonary congestion and dilatation of the left auricle. As the narrowing of the valve orifice increases and the pressures within the pulmonary capillaries and the left auricle mount, there are attacks of pulmonary oedema. Further stenosis limits the exercise tolerance progressively, and may be associated with hemoptysis. Myo-

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cardial dilatation and damage occur, causing arrhythmias. In the terminal stages of the disease, death results from cardiac failure.

Specific patho-physiological phenomena are responsible for this downward clinical course of the patient (Fig. 1). The normal mitral valve has a patent orifice that is 4–6 sq. cm. in area. As valve size is reduced by fibrosis and contraction, the pressure within the left auricle begins to rise, and there is an increased pulmonary capillary pressure. When the pressure within the pulmonary capillary bed is of the order of 20 mm. Hg., intimal proliferation and medial hypertrophy of the pulmonary arterioles occur and result in an increased pulmonary vascular resistance. Increased pulmonary vascular resistance produces a rise in pulmonary arteriolar pressure, which in turn leads to right ventricular

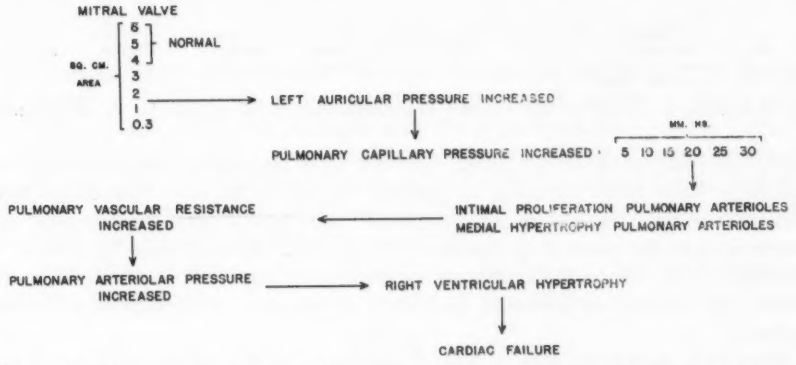

FIG. 1. A schematic interpretation of the sequence of pathologic physiology in mitral stenosis.

dilatation and hypertrophy. The end result of this chain of physiological changes is, of course, cardiac failure. It should be emphasized that right ventricular hypertrophy and the cardiac failure which may follow occur only after pulmonary vascular resistance has increased, and is accompanied by intimal proliferation and medial hypertrophy of the pulmonary arterioles (6).

THE SELECTION OF PATIENTS FOR MITRAL COMMISSUROTOMY

In the natural history of relatively pure mitral stenosis, four distinct stages of the disease can be defined clinically (7) and are employed as a basis to facilitate the selection of patients for surgery (Fig. 2). Valve size cannot always be correlated with the severity of disease, and it is further obvious that there may be considerable overlap between stages of the disease in the individual patient. Nevertheless, these facts do not obviate the utility of the classification.

Patients in Stage I have a mitral valve area in excess of 1.2 sq. cm., and their pulmonary vascular resistances are normal. Therefore, right ventricular dilatation and hypertrophy are not present. The left auricle is enlarged, however, and dyspnoea occurs on violent exertion. Surgery seldom has been performed on this group of patients in the past, and the feasibility of operation remains a most debatable subject. There is an increasing tendency on the part of certain cardiolo-

STAGE	SYMPTOMATOLOGY	VALVE AREA SQ. CM.	AURICLE ENLARGED (X-RAY)	PULMONARY VASCULAR RESISTANCE	RIGHT VENTRICULAR HYPERTROPHY (ECG)	SURGERY	MORTALITY
I	DYSPNEA ON VIOLENT EXERTION	> 1.2	YES	NORMAL	NO	DEBATABLE	VERY LOW
II	SEVERE—PULMONARY DYSPNEA ON ANY EFFORT, ORTHOPNEA PULMONARY EDEMA HEMOPTYSES	1.2	YES	LITTLE INCREASE	+ OR −	IDEAL	3-4 %
III	DYSPNEA, FATIGUE, CURTAILED ACTIVITY	1.0 OR LESS	YES	6–10 TIMES NORMAL	YES	INDICATED	3-4 %
IV	TERMINAL BED-RIDDEN, ALWAYS ON VERGE OF PULMONARY EDEMA	< 0.8	VERY	> 10 TIMES NORMAL	MARKED	TRANSIENT RELIEF	23 %

FIG. 2. Criteria employed for the selection of patients with mitral stenosis for commissurotomy.

gists, however, towards the belief that the most impressive results of commissurotomy have been obtained in patients subjected to operative intervention *prior* to the development of intimal proliferation and medial hypertrophy, and hence prior to the onset of an increased vascular resistance within the pulmonary circulation (8). Since the operative mortality in Stage I is extremely low, the future may witness an increased incidence of operative interventions with these patients.

Stage II is associated with a critical narrowing of the mitral valve to an area of 1.2 sq. cm., and is characterized by both left auricular hypertrophy and a slight increase in pulmonary vascular resistance. There may or may not be electrocardiographic or radiologic evidence of right ventricular dilatation and hypertrophy. Patients in this group have severe pulmonary symptoms on almost any exertion, since the output of the left ventricle is relatively fixed and any marked increase in output from the right ventricle must result in an increased pressure within the left auricle and the pulmonary bed, with the occurrence of orthopnoea, paroxysmal nocturnal dyspnoea, attacks of acute pulmonary oedema, and hemoptyses. These patients are ideal candidates for surgical intervention, and the mortality rate in this and the following group (Stage III) is generally only about 3–4 per cent.

The valve area in Stage III is 1.0 sq. cm. or less, and the pulmonary vascular resistance is six to ten times the normal. There is evidence of left auricular, right ventricular, and pulmonary artery hypertrophy and dilatation; and the electrocardiogram also demonstrates marked right ventricular hypertrophy. The patients in this group have marked curtailment of activity: they are constantly tired and fatigued, and may remain at total rest so constantly that dyspnoea as a symptom is not even evident. However, the slightest exertion will produce the entire gamut of pulmonary symptomatology. Surgery is most definitely indicated for these patients; the mortality rate of operation is about 3–4 per cent.

Patients in Stage IV have a valve area that is less than 0.8 sq. cm., and a pul-

monary vascular resistance that is more than ten times the normal. Cardiac enlargement of up to 50 per cent is found in these patients: there is a huge right ventricle, prominent pulmonary artery, increased pulmonary vascular markings, and a greatly enlarged left auricle, demonstrable on radiologic examination; and there is definite electrocardiographic evidence of right ventricular hypertrophy. These patients are in the terminal stages of the disease, and are frequently in right-sided heart failure: they are bed-ridden, cachectic, and completely incapacitated, being constantly on the verge of pulmonary oedema. Surgery for this group is extremely hazardous, the mortality rate running 23 per cent. The results are occasionally dramatic in those who survive the operative intervention, but the relief is apt to be transient owning to the irreversible cardiac and pulmonary damage.

The Contra-Indications to Mitral Commissurotomy

The selection of those patients with mitral disease for whom surgical therapy is indicated is without doubt the most important single aspect of mitral commissurotomy; and while this decision must be a co-operative venture between cardiologist, surgeon, and anaesthesiologist, it is obviously the cardiologist who must bear the brunt of the burden. The difficulties are increased by the fact that there are certain complicating factors which must also be taken into consideration in the selection of candidates for surgery.

At least three of these conditions must be regarded as contra-indications to mitral commissurotomy (Table I). Activity of the rheumatic process is quite

TABLE I

The Contra-Indications to Mitral Commissurotomy

I. *Absolute Contra-Indications*
- A. Acute rheumatic activity
- B. Acute or subacute bacterial endocarditis
- C. Significant involvement of the left ventricle
  1. Aortic stenosis
  2. Aortic regurgitation
  3. Mitral regurgitation

II. *Relative Contra-Indications*
- A. Calcification of mitral valve (disappointing result)
- B. Pregnancy (abortion best; if operation necessary, between 16th and 28th week)
- C. Cardiac failure
- D. Pulmonary infarction (anticoagulant therapy prior to commissurotomy)

uncommon at the late stage of the disease process characterized by a critical narrowing of the mitral orifice, but when it is present it constitutes an absolute contra-indication to operation. Similarly, acute and subacute bacterial endocarditis are rare in patients being evaluated for possible surgical therapy, but also are an absolute contra-indication to operation when present. Finally, evidence of a significant involvement of the left ventricle, indicating the superimposition of the effects of other valvular lesions upon the mitral stenosis, must be considered a third absolute contra-indication to surgical intervention. This is

not to say that only "pure" mitral stenosis warrants surgery; but, rather, that mitral commissurotomy may enhance the undesirable effects of other valvular disease in certain mixed lesions and thus leave the patient more crippled than prior to the operation. The evaluation of the operability of multivalvular lesions is difficult, the decision being dependent upon whether or not the mitral stenosis is the limiting or dominant lesion physiologically. Lesions of the aortic valve, both stenosis and insufficiency, occur commonly in conjunction with mitral stenosis. If other lesions are of sufficient physiologic importance to produce left ventricular enlargement or damage, then surgical correction of a coexisting mitral stenosis is not feasible. Similarly, patients with severe mitral regurgitation, when it predominates the mitral stenosis, will not benefit from the operation of mitral commissurotomy.

There are, in addition, several conditions which might be termed relative contra-indications to surgical intervention (Table I), but which are in reality only complicating factors that must be taken into consideration in the evaluation of patients for commissurotomy rather than contra-indications to operation. Calcification of the mitral valve, for instance, when extreme, may be associated with a disappointing surgical result owing to a failure of the mitral orifice to remain patent even after an adequate fracture of the commissures; but it is not a true contra-indication to operation. Pregnancy is a second example of a complicating factor that nevertheless does not constitute a true contra-indication to surgery: the greatly increased load which is placed upon the cardiovascular system, particularly during the final trimester, adds an extra risk to mitral disease (9); when the termination of pregnancy is unacceptable to the patient but parturition threatens catastrophe, mitral commissurotomy is performed between the 16th and 28th weeks. Frank cardiac failure cannot be regarded as a contra-indication to operation either, but its presence most certainly does increase the difficulties in an already complex situation. Finally, the occurrence of recent pulmonary infarction, while also not a contra-indication to commissurotomy, may warrant anticoagulant therapy prior to surgical intervention.

The Anaesthetic Requirements of Mitral Commissurotomy

Knowledge of the hemodynamic basis of the clinical syndromes and the physiological deviations encountered in patients with mitral stenosis permits an enumeration of the factors which must be considered in the development of any anaesthetic technique designed for the management of the patient undergoing mitral commissurotomy (Table II).

A major tenet of the anaesthetic management must be the avoidance of any physical effort by the patient. Slight exertion may precipitate the onset or progression of frank cardiac decompensation in the patient with mitral stenosis. Such exertion during anaesthesia may take several forms: a tumultuous second stage of anaesthesia during induction; "bucking" against the endotracheal catheter during intubation, when there is too light a plane of anaesthesia following intubation (particularly during certain surgical stimulations or during tracheobronchial suction), or at the time of extubation; straining during the course of operation itself; or a period of restlessness and even delerium during emergence

TABLE II

THE ANAESTHETIC REQUIREMENTS OF MITRAL COMMISSUROTOMY

1. Rapid, quiet induction
2. Adequate oxygenation—patent airway, high flows O_2, positive pressure
3. Avoidance of exertion—straining or "bucking"
4. Maintenance of normotension—throughout operation
5. Lightest possible level of anaesthesia
6. Non-toxic anaesthetic agents—not irritable to myocardium
7. Non-explosive anaesthetic agents—if cautery to be employed
8. Control of cardiac arrhythmias
9. Rapid, quiet emergence

or in the immediate postoperative period. All these several forms of exertion must be avoided by the employment of certain appropriate anaesthetic manoeuvres.

A second major tenet is the maintenance of adequate oxygenation, particularly of the myocardium, throughout the operative and postoperative periods. This is accomplished by ensuring a completely patent airway, by employing high flows of oxygen, and by a sufficient use of positive pressure during respiration both to overcome the constant tendency towards pulmonary oedema and "drive" the oxygen across the alveolar membrane to the pulmonary vascular bed.

Normal systemic blood pressure must be preserved both during induction and throughout the operative intervention, inasmuch as oxygenation of the myocardium is dependent upon mean aortic pressure (10). Anaesthetic agents that would depress myocardinal contractility and thus depress blood pressure must therefore not be employed. Normal peripheral resistance is particularly important, since the output of the left ventricle is relatively fixed and cannot increase to compensate for an enlarged capacity of the peripheral vascular bed. Therefore, the vasodilatation which follows upon depression of the medullary centres with even moderate depths of anaesthesia must be avoided by the employment of the lightest possible level of general anaesthesia. The hypotension that often accompanies actual fracture of the valve almost invariably reverts to normotension following the cessation of this particular surgical manipulation.

It is essential also that the diseased myocardium be protected from either physical or chemical stimulations that would tend to induce irritability and severe cardiac arrhythmias. Towards this end, non-toxic anaesthetic agents must be employed; and steps must be taken to decrease the irritability of the myocardium, or to control serious arrhythmias when they do occur, without at the same time producing a severe depression of that muscle.

Finally, it is important to employ non-explosive anaesthetic agents in order to permit the use of electro-coagulation, electrocardiographic recorder with a hot-stylus writing point, or other electrical equipment that might be a fire or explosion hazard.

THE ANAESTHETIC TECHNIQUE FOR MITRAL COMMISSUROTOMY

These multiple requirements have imposed stringent limitations upon the anaesthetic techniques employed for the surgical therapy of mitral stenosis. It is essential that the lightest possible level of general anaesthesia be maintained in

order to obviate the development of hypotension; yet, at the same time, there must be sufficient anaesthetic control over the patient to prevent any "bucking," straining, or exertion. This seeming paradox has been resolved in the present technique by the use of nitrous oxide–oxygen anaesthesia in combination with a dilute intravenous infusion of succinylcholine.

Nitrous oxide, when administered with an abundant concentration of oxygen, is perhaps the least toxic of all the general anaesthetic agents and at the same time the most analgesic. It has the single disadvantage for purposes of commissurotomy that it produces levels of anaesthesia that are so light that surgical manipulations within the thorax and the presence of an endotracheal tube within the trachea are but poorly tolerated without "bucking," straining, or other exertion. This disadvantage can be overcome by the use of succinylcholine, itself a non-toxic drug, to provide graded muscular relaxation that is controllable on a minute-to-minute basis. The combination of nitrous oxide–oxygen and succinylcholine produces an extremely light degree of general anaesthesia and yet prevents straining or exertion on the part of the patient. When, in addition, small doses of pentothal are employed for induction, intermittent doses of demerol are injected intravenously to maintain basal narcosis, and myocardial depressants are administered judiciously to control hyper-irritability of the myocardium, a form of modern balanced anaesthesia results which serves admirably for the anaesthetic management of the patient undergoing mitral commissurotomy (Table III).

TABLE III

ANAESTHETIC TECHNIQUE FOR MITRAL COMMISSUROTOMY

1. Premedication—little or no parasympatholytic drug
2. Induction—pentothal, to loss of eyelid reflex
3. Inhalation—N_2O:O_2 (6:2 litres), semi-closed technique
4. Intubation—apnoeic technique: succinylcholine 0.2 per cent infusion
5. Positioned carefully and slowly
6. Maintenance—

   N_2O:O_2 (6:2 litres)
   Succinylcholine 0.2 per cent infusion (assisted respirations)
   Demerol 10 mgm. i.v. (p.r.n.)

7. Periodic reinflation of lung
8. Tracheal suction only when absolutely necessary
9. Extubation—apnoeic technique: succinylcholine 0.2 per cent infusion
10. Moved to bed slowly, carefully, and only after an interval for stabilization

Premedication. The premedication must differ in certain important respects from that employed in the ordinary surgical patient. Tachycardias are the most frequent and most serious disturbances of rhythm encountered during operation upon the patient with mitral stenosis: filling of the left ventricle is restricted by the stenotic mitral valve, and a superimposed tachycardia will reduce the amount of blood reaching the ventricle. The parasympatholytic drugs are therefore omitted from the preanaesthetic medication because of the danger that even moderate degrees of vagal blockade may precipitate this undesirable arrhythmia.

The usual sedative and hypnotic drugs are employed in light to moderate

dosage, for the patient is frequently so anxious to have the operation and the relief that it promises that apprehension is a minor or even an absent problem. This happy state of affairs does not exist on all occasions, however, and it is important to note that preoperative tachycardia and even pulmonary oedema may be precipitated by apprehension (11). A middle course must be maintained, therefore, and premedication consists of 100 mgm. of demerol administered hypodermically one hour prior to induction, with the addition of pentobarbital 100 mgm. by mouth two hours prior to induction for the very robust or excessively nervous patient.

Induction. Anaesthesia is induced by the intravenous administration of small (2-3 cc.), repeated doses of 2½ per cent pentothal. The circulation time may be distinctly prolonged in the patient with mitral stenosis, and almost a minute must be allowed before the effects of the pentothal are judged and reinjection is performed. The depressant effects of pentothal upon the myocardium, with the subsequent production of hypotension, are very real: the great advantages of a rapid, quiet, smooth induction with pentothal must be achieved, therefore, with due consideration to slow injections of minimal amounts of this agent.

Inhalation of nitrous oxide–oxygen, 6 litres: 2 litres, is begun by the semi-closed circle carbon dioxide absorption technique following the loss of the eyelid reflex, which occurs generally after a total dosage of 100-250 mgm. of pentothal.

Endotracheal intubation. When the induction of anaesthesia has been completed, a dilute, 0.2 per cent concentration of succinylcholine in 5 per cent glucose water is started as an intravenous infusion and continued until total apnoea supervenes. During the onset of muscular relaxation, respirations are assisted; when total muscular relaxation and apnoea occur, respirations are controlled. With the onset of apnoea, the patient is hyperventilated with the nitrous oxide–oxygen mixture and then endotracheal intubation is performed, employing the largest, cuffed catheter that will fit through the glottis easily. Prior to intubation, the tip of the endotracheal catheter is lubricated with a water-soluble 1 per cent metycaine anaesthetic jelly, to provide anaesthesia of the vocal cords of sufficient duration to persist throughout operation, but short enough to permit an active cough reflex at the termination of operation or soon thereafter.

Positioning patient for operation. The patient is then positioned carefully and slowly for left thoracotomy. It is essential that this be done in such a manner that sudden shifts of posture, which can lead to profound falls in the blood pressure of the anaesthetized patient, are avoided. Apnoea is maintained by a continuation of the succinylcholine infusion throughout this manoeuvre of posturing in order to prevent "bucking" upon the endotracheal catheter.

Maintenance. Maintenance anaesthesia is established after the patient has been properly postured for operation, and differs from that employed during induction and intubation in two minor, but very important, particulars.

First, the infusion rate of succinylcholine is decreased very gradually to permit the return of spontaneous respiratory activity and the substitution of assisted respirations for controlled respirations. The use of positive pressure during late inspiration is desirable both to offset the possibility of pulmonary oedema and to aid in driving oxygen across the pulmonary membrane. It is important to avoid

the use of continuous controlled respirations in the patient with mitral stenosis, however, since a constant intrapulmonary positive pressure will introduce an added resistance to the flow of blood through the pulmonary circulation and will place a further burden upon the overworked right ventricle. Furthermore, in the technique of continuous succinylcholine infusion, apnoea must be avoided assiduously, for a slight degree of respiratory activity provides the only evidence as to the required rate of flow of the succinylcholine infusion. If apnoea and controlled respirations are maintained throughout operation by the infusion of succinylcholine, it is quite possible to administer larger amounts of succinylcholine than the body can metabolize, and a prolonged period of postoperative apnoea may result. The great majority of the instances of prolonged apnoea which have been reported following the uses of succinylcholine have undoubtedly occurred on this basis (12). It is also important to effect the transition from controlled to assisted respirations very gradually, for a sudden diminution in the rate of flow of succinylcholine may allow a sudden resumption of a marked degree of muscular activity; since the patient is being maintained in an extremely light plane of nitrous oxide–oxygen (6 liters : 2 liters) anaesthesia, the return of marked muscular activity may be associated with dangerous "bucking" or straining.

Second, small (10-20 mgm.) intravenous doses of demerol are administered at intervals to supplement the degree of basal narcosis (Fig. 3). The addition of demerol to the combination of succinylcholine–nitrous oxide produces slightly more extensive anaesthesia, without at the same time inducing deep levels of general anaesthesia, than can be maintained by nitrous oxide alone. This extra degree of anaesthesia aids both in preventing the occurrence of "bucking" and, perhaps, in obtunding autonomic activity in these lightly anaesthetized patients. The use of demerol has the added advantages of exerting some quinidine-like action upon the heart and of producing a certain amount of postoperative analgesia and sedation. The total dosage of demerol employed throughout operation is not large, generally less than 200 mgm. depending upon the individual patient and the duration of the surgical intervention. The indications for the administration of these small, intermittent doses of demerol are somewhat nebulous and difficult of definition: one, two, or even three doses of 20 mgm. each are administered soon after the patient is postured and maintainenance anaesthesia is begun; subsequent 10 mgm. doses are injected in response to a slight urgency of inspiration as detected by the anaesthesiologist's hand on the rebreathing bag.

Tracheal suction and reinflation of lung. The problem of whether or not tracheal suction should be employed, and if so, how often, remains an enigma. The tracheo-bronchial tree must certainly be cleared if adequate oxygenation is compromised by the presence of accumulated fluids. On the other hand, pulmonary oedema is a constant and a dreaded threat, and its occurrence may be prompted by the production of strong negative intrapulmonary pressure. A middle course must be steered, and tracheal suction is employed only when urgently necessary. Reinflation of the collapsed lung is carried out periodically, every 20 to 30 minutes, in the same manner as during any other type of thoracotomy.

Endotracheal extubation. Endotracheal extubation is performed with the same anaesthetic technique as that employed for intubation, with the patient in the

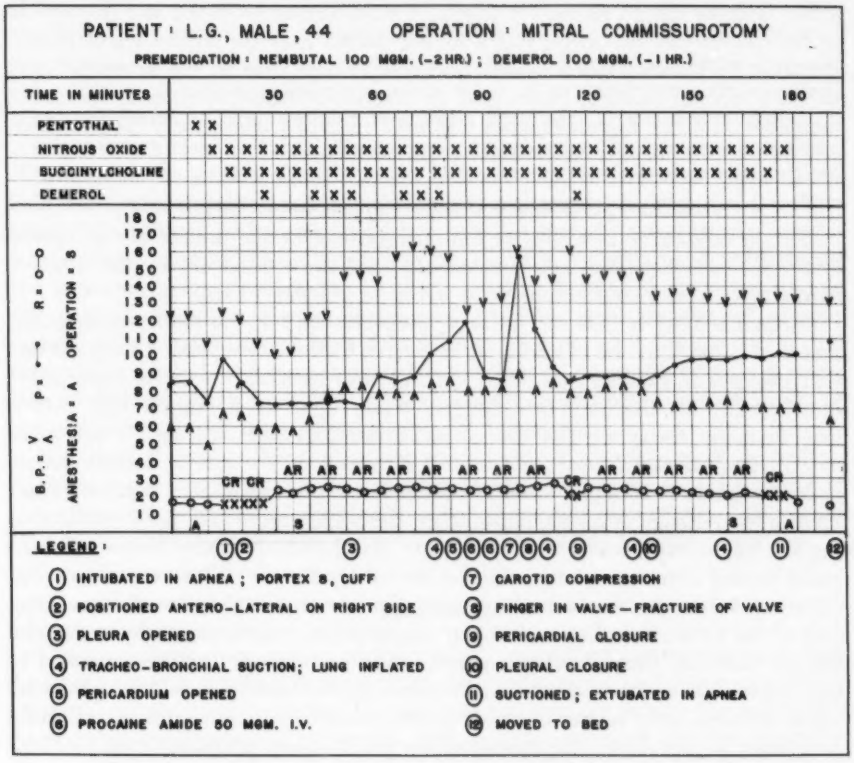

FIG. 3. Anaesthetic chart of patient undergoing mitral commissurotomy.

apnoeic state, in order to prevent the occurrence of "bucking" or straining. At the termination of operation, and following the application of dressings to the wound, the rate of flow of the succinylcholine infusion is increased to reproduce apnoea and controlled respirations replace the assisted respirations that were employed throughout the maintenance of anaesthesia. The pharynx is suctioned thoroughly, the lungs inflated several times with 100 per cent oxygen, the cuff on the endotracheal tube is deflated, and the endotracheal catheter is removed. Both the succinylcholine infusion and the inhalation of nitrous oxide are discontinued, and controlled respirations with 100 per cent oxygen are employed until spontaneous respiratory activity resumes. Assisted respirations with 100 per cent oxygen are continued further until *full* respiratory activity is present. This generally occurs within a matter of 3-9 minutes, but it is absolutely imperative that assisted respirations with 100 per cent oxygen be continued until there is no doubt as to the total efficiency of the respiratory exchange.

Post-operative care. Following the resumption of total respiratory activity and an interval of time for stabilization, the patient is moved slowly and carefully into bed. There must be no hurry to remove the patient from the operating table;

a few minutes should suffice for adequate stabilization following the resumption of full respiratory exchange, but a longer period may be required and should certainly be allowed when necessary. Postoperative care in the recovery room includes oxygen therapy, pharyngeal or tracheo-bronchial suction as indicated, moderate sedation, and meticulous and ever vigilant nursing care. Medical therapy during this phase is supervised by the cardiologist.

Adjunctive Measures during Mitral Commissurotomy

Certain adjunctive measures to the management of the patient are of utmost importance during mitral commissurotomy, and are carried out by the surgeon, cardiologist, and anaesthesiologist working in concert (Table IV).

Cardiac activity is followed on an appropriate lead of the electrocardiograph, and is monitored by the cardiologist in person. Certain experienced surgeons feel that changes in cardiac tone, rate, rhythm, and irritability can be appreciated most readily by the surgeon's fingers. While there is no doubt that this is true in many instances, the use of electrocardiographic tracings is advisable as a further check to ensure the correct diagnosis of changes in cardiac action. Procaine amide in 50 mgm. doses is administered intravenously in repeated doses up to a total dosage of 50-300 mgm. when the myocardium is hyper-irritable during the initial exposure of the left auricle. Sinus tachycardia, a frequent occurrence during commissurotomy, may be slowed by the use of intravenous prostigmine in 0.5-3.0 mgm. doses. Multiple arrhythmias occur at the time of actual fracture of the valve, including ventricular extrasystoles, ventricular tachycardia, and bizarre patterns; these, however, require no other therapy than the removal of the surgeon's finger from the valve. Atropine may be employed in doses of 0.3-1.2 mgm. intravenously in the event of severe bradycardia; moderate bradycardia frequently occurs during the closure of the pericardium, presumably on the basis of vagal stimulation, but seldom requires the institution of active therapeutic measures.

The use of vasopressor agents to combat hypotension has been a moot question. Moderately profound hypotension frequently attends upon the actual fracture

TABLE IV

Adjunctive Measures during Mitral Commissurotomy

1. ECG monitor of cardiac events.
2. Control of cardiac arrhythmias
   - A. Procaine amide: 50–300 mgm. i.v.
   - B. Prostigmine: 0.5–3.0 mgm. i.v.
   - C. Atropine sulphate: 0.3–1.2 mgm. i.v.
3. Vasopressor agents—if prolonged or persistent hypotension
4. Blood and fluid replacement—little necessary or desirable, but must be prepared for possible surgical exigency
   - A. 15 gauge cannula *in situ*, and slow intravenous drip
   - B. Compatible blood for transfusion at hand
   - C. Blood pump available
   - D. Intra-arterial transfusion set-up ready
5. Compression of carotids at time of valve fracture—if history of auricular fibrillation or previous embolism

of the mitral valve, since all flow of blood to the left ventricle is terminated temporarily at this point in the operation; normotension, or at least lesser degrees of hypotension, generally follows promptly upon the removal of the finger from the valve orifice. A potent vasopressor agent, such as neosynephrine or levophed, however, should be kept available to combat persistent, severe hypotension.

Blood transfusions and fluid replacement are seldom either necessary or desirable. Indeed, the greatest danger is that unnecessary transfusions or over-zealous fluid therapy may induce congestive failure or pulmonary oedema. On the other hand, it is essential to be prepared for a possible surgical exigency, for a gross haemorrhage of considerable magnitude can occur in a matter of seconds should the auricular opening escape the surgeon's control. A large intravenous cannula (15 gauge), therefore, should be inserted prior to operation and its patency maintained during operation by a slow infusion of 5 per cent glucose and water; compatible whole blood is kept available for transfusion in the operating room. It is advisable to have a mechanical device for pumping blood intravenously, or even an intra-arterial transfusion set-up (2), in readiness.

Finally, if there has been a history of auricular fibrillation or of embolism, bilateral carotid compression is performed at the time of the actual fracture of the valve. While the anaesthesiologist can perform this manoeuvre, it is preferable to have the duty devolve upon some other available medical personnel.

Summary

The anaesthetic management of the patient undergoing commissurotomy must be based upon a detailed knowledge of the fundamental pathologic physiology of mitral stenosis. As increasing fibrosis and constriction reduce the valve area towards the critical size of 1.2 sq. cm., left auricular pressure rises and produces an increased pulmonary capillary pressure. The latter induces intimal proliferation and medial hypertrophy of the pulmonary arterioles, causing increased pulmonary vascular resistance. Right ventricular dilatation and hypertrophy follow, setting the stage for cardiac failure.

Mitral disease can be classified into four clinical syndromes, or stages, which serve as a basis for the selection of patients for surgery. Stage I is characterized by an uncritical narrowing of the mitral valve, left auricular dilatation and hypertrophy, and dyspnoea on exertion: these patients have not usually been regarded as candidates for commissurotomy in the past. Stage II is associated with a mitral valve size of about 1.2 sq. cm., both left auricular and right ventricular hypertrophy, and severe pulmonary symptomatology upon almost any exertion: these patients are ideal candidates for the operation. Stage III is accompanied by a valve size of less than 1.0 sq. cm., greatly increased pulmonary vascular resistance and marked right ventricular hypertrophy, and symptoms of constant fatigue even at total rest: surgical intervention is definitely indicated in these pulmonary cripples, and its results are often dramatic. Patients in Stage IV have a mitral valve area less than 0.8 sq. cm., cardiac enlargement, and are terminal, bed-ridden and cachectic: surgery is extremely hazardous, and while the results are occasionally miraculous, the relief is often transient.

Subacute bacterial endocarditis, rheumatic activity, and predominant mitral regurgitation, aortic stenosis or aortic regurgitation in mixed lesions, must be considered absolute contra-indications to surgery. Marked calcification of the mitral valve, pregnancy, frank cardiac failure, and recent embolism are not true contra-indications, but are complicating factors necessitating special considerations.

The anaesthetic requirements of mitral commissurotomy include a rapid, quiet induction; adequate oxygenation at all times; avoidance of any exertion, straining, or "bucking"; maintenance of normotension throughout operation; the lightest possible level of general anaesthesia; the use of non-toxic and non-explosive anaesthetic agents; the control of cardiac arrhythmias; and a rapid, quiet emergence from the anaesthetic state.

The anaesthetic technique is based upon the use of succinylcholine–nitrous oxide anaesthesia for the fulfilment of these varied requirements. Premedication is light or moderate, and parasympatholytic drugs are avoided. Small, repeated doses of pentothal are employed for induction, following which nitrous oxide–oxygen, 6 liters : 2 liters, is administered by the semi-closed circle carbon dioxide absorption technique. Succinylcholine, 0.2 per cent concentration in 5 per cent glucose in water, is begun as a constant intravenous infusion and continued until apnoea occurs; at this time, oral intubation is performed with a large, cuffed endotracheal catheter. After slow and careful posturing of the patient, the succinylcholine infusion is slowed until spontaneous respiratory activity is just evident. Anaesthesia is then continued with the nitrous oxide–oxygen mixture and assisted respirations throughout, the flow rate of the succinylcholine infusion being adjusted constantly to maintain minimal, but spontaneous, respiratory activity. Small (10-20 mgm.) doses of demerol are administered intravenously at intervals to supplement the basal narcosis. The lungs are reinflated periodically, but tracheal suction is avoided except when the gross accumulation of fluids in the tracheo-bronchial tree make its use mandatory. At the termination of operation, the succinylcholine infusion rate is again increased to produce apnoea for the removal of the endotracheal catheter. An interval for stabilization is permitted after the cessation of the succinylcholine infusion and the resumption of *complete* respiratory activity before the patient is moved, slowly and carefully, into bed.

Cardiac activity is monitored continuously by the electrocardiograph. Procaine amide, in 50-300 mgm. doses, is injected intravenously to control any hyperirritability of the myocardium during the exposure of the left auricle. Sinus tachycardia may be slowed by the use of intravenous prostigmine in 0.5-3.0 mgm. doses. Bradycardia rarely becomes a problem, but may be an indication for the intravenous administration of 0.3-1.2 mgm. of atropine sulphate. Prolonged and profound hypotension may necessitate the use of vasopressor agents. Fluid therapy is restricted at all times, in order to prevent overloading the circulation; but compatible blood and a pressure transfusion set-up should be available in the event of a surgical exigency leading to gross haemorrhage. Bilateral carotid compression is employed if there has been auricular fibrillation or previous embolism.

Résumé

Le traitement anesthésique du malade qui subit une commissurotomie doit reposer sur une connaissance détaillée de la physiologie pathologique fondamentale de la sténose mitrale. La fibrose et la constriction, quand elles augmentent, réduisent d'autant la surface de la valvule jusqu'à atteindre la dimension critique de 1,2 cm^2, la pression monte dans l'oreillette gauche et provoque une augmentation de la pression capillaire pulmonaire. Cette dernière amène une prolifération de l'intima et une hypertrophie de la média des artérioles pulmonaires, produisant ainsi un accroissement de la résistance vasculaire pulmonaire. Une dilatation et une hypertrophie du ventricule droit s'ensuivent, préparant le terrain à l'insuffisance cardiaque.

Les affections mitrales peuvent se classer en quatre syndrômes, ou stades, cliniques, qui servent de base à la sélection des malades à opérer. Le premier stade est caractérisé par un rétrécissement non-critique de la valvule mitrale, une dilatation et hypertrophie de l'oreillette gauche, de la dyspnée d'effort: d'habitude, ces malades n'ont pas été dans le passé considérés comme candidats à la commissurotomie. Le deuxième stade se caractérise par une valvule mitrale d'environ 1,2 cm^2 de surface, une hypertrophie à la fois auriculaire gauche et ventriculaire droite, et une symptomatologie pulmonaire sérieuse après le plus petit effort: ces malades sont les candidats parfaits à l'opération. Le troisième stade s'accompagne d'une valvule mitrale de moins de 1 cm^2 de surface, d'une augmentation considérable de la résistance vasculaire pulmonaire, d'une hypertrophie ventriculaire droite marquée et de symptômes constants de fatigue, même au repos absolu: l'intervention chirurgicale est toute indiquée dans ces conditions pulmonaires critiques et les résultats sont souvent spectaculaires. Les malades du quatrième stade ont une surface de la valvule mitrale inférieure à 0,8 cm^2, un grossissement cardiaque et sont dans la phase finale de la maladie, cloués au lit et cachectiques: la chirurgie est extrèmement hasardeuse, et bien que les résultats tiennent quelquefois du miracle, le soulagement est souvent passager.

L'endocardite bactérielle subaigüe, l'activité rhumatismale, ainsi que l'insuffisance mitrale prédominante, la sténose aortique ou l'insuffisance aortique compliquées de lésions, doivent être considérées comme contre-indications absolues de la chirurgie. Les calcifications nettes de la valvule mitrale, la grossesse, l'insuffisance cardiaque marquée, et les embolies récentes ne sont pas exactement des contre-indications mais des facteurs de complication qui exigent des considérations spéciales.

L'anesthésie pour la commissurotomie mitrale exige: une induction rapide et douce; une oxygénation constamment adéquate; l'absence de tout effort, tension ou "cabrage"; maintien de la tension normale pendant toute l'opération; le niveau le plus léger possible d'anesthésie générale; l'emploi d'agents anesthésiques non-toxiques et non-explosifs; le contrôle des arythmies cardiaques; et une sortie rapide et douce de l'état anesthésique.

La technique anesthésique est basée sur l'emploi de l'anesthésie au protoxyde d'azote-succinylcholine, en vue de satisfaire à ces diverses exigences. Le prémédication est légère ou modérée, et on évite les médications parasympatholytiques.

Pour provoquer le sommeil, on emploie de petites doses répétées de pentothal, en suite de quoi on administre du protoxyde d'azote-oxygène dans la proportion 6 litres/2 litres par la technique de l'absorption de l'anhydride carbonique en circuit demi-fermé. On commence l'injection intra-veineuse continue, de la succinylcholine, sous la forme d'une concentration à 0,2% dans 5% de glucose dans l'eau et on continue jusqu'à apparition de l'apnée; à ce moment, on pratique une intubation orale à l'aide d'une large sonde endotrachéale munie d'un brassard pneumatique. Après avoir mis le malade en position, lentement et avec soin, on ralentit l'injection de succinylcholine jusqu'à ce que l'activité respiratoire spontanée soit tout juste évidente. On continue alors l'anesthésie avec le mélange protoxyde d'azote-oxygène et avec des respirations assistées tout au long, le débit de l'injection de succinylcholine étant constamment réglé de manière à maintenir une activité respiratoire minime, mais spontanée. On administre en intra-veineuses de petites (10-20 mg) doses de démerol de temps à autre pour compléter l'anesthésie de base. On insuffle périodiquement les poumons, mais on évite les succions trachéales sauf quand l'accumulation de mucosités dans l'arbre trachéo-bronchique en rend l'emploi impératif. A l'issue de l'opération, le débit de l'injection de succinylcholine est à nouveau accru pour produire l'apnée pendant l'extraction de la sonde endotrachéale. On ménage un intervalle après la cessation de l'injection de succinylcholine et la reprise *complète* de l'activité respiratoire avant de remettre, lentement et avec soin, le malade au lit.

L'activité cardiaque est suivie en permanence sur l'électrocardiographe. On injecte, en intra-veineuse, de la procaine amide, en doses de 50 à 300 mg pour pallier à toute hyper-irritabilité du myocarde pendant la mise à nu de l'oreillette gauche. La tachycardie des sinus peut être ralentie par l'emploi de prostigmine en doses de 0,5 à 3 mg intraveineuses. La bradycardie devient rarement un problème mais peut être une indication de l'administration intraveineuse de 0,3 à 1,2 mg de sulfate d'atropine. Une hypotension profonde et prolongée peut exiger l'emploi d'agents vasoconstricteurs. On évite à tout moment la rehydration thérapeutique, pour éviter de surcharger la circulation; mais il est bon d'avoir sous la main du sang compatible et un appareil de transfusion sous-pression pour prévenir tout besoin chirurgical entrainant une grosse hémoragie. On emploie la compression bilatérale de la carotide s'il y a eu fibrillation auriculaire ou embolie antérieure.

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THE ANAESTHETIST AND PULMONARY TUBERCULOSIS

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In this paper, we are going to describe the organization of an Anaesthesiologic Department in a modern sanatorium where major chest surgery is done for tuberculosis patients; the description will include the role played by the anaesthetist during the pre- per- and post-operative periods.

The Preoperative Period

As many of them represent poor anaesthetic risks, the importance of a very attentive preparation on these pulmonary cases will be realized. We routinely request the following examinations:

1. Clinical findings and physical examination.
2. Laboratory examinations.
3. X-ray.
4. Some cardio-pulmonary function test:
   - (*a*) Vital capacity
   - (*b*) Timed vital capacity
   - (*c*) Maximum breathing capacity
   - (*d*) Ventilation equivalent
   - (*e*) Minute volume
   - (*f*) Blood gas analysis
   - (*g*) Venous pressure
   - (*h*) Circulation time
   - (*i*) E.K.G.

From the results of these examinations, the anaesthetist may arrange a better preparation for the patient, for whom the following things are carried out by the anaesthetist or a qualified person:

1. Restoration of fluid balance (Fluid and blood transfusion).
2. Aerosol therapy (Streptomycin and Vaponefrin).
3. Postural drainage.
4. Physiotherapy.
5. Preoperative sedation.

Our patients under treatment with isonicotinic acid derivatives, especially the isopropyl derivative (Marsilid[1]), have shown oxygenation problems and convulsions at the anaesthetic period. We now routinely stop this medication one month before the operation in order to avoid such complications.

The Preoperative Period

With the best anaesthetic technique, these patients present many problems:

1. Reduction of static lung volume
   - (*a*) Reduction of usable vital capacity

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(*b*) Increase of residual capacity
(*c*) Increase of functional residual air.

2. Inadequate dynamic lung volume
   (*a*) Reduction of tidal air
   (*b*) Reduction of maximum capacity
   (*c*) Inadequate minute volume
   (*d*) Abnormal ventilation equivalent.
3. Paradoxical respiration.
4. Mediastinal flutter.
5. Lessening of cardiac output and blood volume.
6. Delay in the venous return.
7. Hypoxemia or anoxemia.
8. Arterial-venous shunt.
9. Reflex problems.
10. Aspiration of secretions and spread of disease problems.

As the thoracic operation is taking place in the cardio-pulmonary area, which is one of the most reflexogenic zones of the body, both the surgical stimulus and the anaesthetic agents are prone to cause hypotension, bradycardia, arrhythmia, or even cardiac arrest.

As a preventive it might be well to mention preoperative topical anaesthesia of the whole tracheo-bronchial tree, intercostal nerve blocks, a good plane of general anaesthesia, good oxygenation of the patient, and sometimes an intravenous injection of a ganglioplegic drug.

Pulmonary tuberculosis patients frequently show a reduction of blood volume and anaemia. Replacement therapy with blood, plasma, or plasma expanders and electrolytes is imperative in order to avoid shock and anoxemia.

Paradoxical respiration and mediastinal flutter are also serious problems; they can be minimized by intubation techniques, controlled or assisted respiration, and the face down position (Overholt).

Anaesthetic agents are a matter of choice. At Laval Hospital, we use intravenous anaesthetic agents, curares combined with nitrous-oxide Oxygen technique. For one reason or another, after a satisfactory understanding with the surgeons, cyclopropane or ether techniques are also used for some special cases.

Recently, for poor-risk patients, we tried a new technique of sedation of the autonomic nervous system devised in France by Huguenard and Laborit with "Cocktailytique": Demerol, Diparcol, Phenergan, Largactil, associated or not with local or regional anaesthesia.

For intubation, endobronchial anaesthesia with Carlen's tube is our first choice; the advantages of this technique have been summarized as follows, by Dr. Carlens in *Anesthesiology*, January 1953 issue:

1. Once the chest is open, the diseased lung may be deflated to provide greater operating space when the lung is separated from the chest wall.

2. The lung can be inflated when lobar or intersegmental planes are developed.

3. The bronchus can be divided, whenever this is suitable during the resection and left open until it is convenient to close it. During the time the bronchus is

open, a small plastic catheter is left in place for continuous suction, with suction close to the opening of the bronchus to collect blood and secretions.

4. It is not necessary to apply a clamp on the proximal part of the bronchus; thus injury to the bronchial wall is avoided.

5. The bronchial tree may be inspected and aspirated by a small rubber catheter through the open bronchus.

6. Bronchotomies and resection of the carina and of the tracheal wall are facilitated by this method of endobronchial anaesthesia.

7. In wet cases during repeated aspirations one lung is always ventilated and the same plane of anaesthesia can be maintained more easily. In some pneumonectomy cases with empyema we use endotracheal or endobronchial tubes along with the Thompson's blocker.

Before the time of Thompson's blocker and Carlens' catheter we had to rely on simple intubation with endotracheal or endobronchial tubes. However, in children's surgery, the tracheo-bronchial tree does not allow any other technique and we still use these simple intubations.

Last year, Dr. Carlens paid a visit to our hospital and told us that five thousand cases were done in Sweden with his catheter with many advantages and without any harmful incident.

Up to now, at Laval Hospital, this technique of bronchial anaesthesia (Carlens' tube) has been used in more than five hundred cases: pulmonary resections, decortications, thoracoplasties. We tend to agree with Dr. Carlens' remarks. This tube is also used at our hospital for bronchospirometry. Its introduction is relatively smooth. Nevertheless a previous bronchoscopic examination is helpful.

The functions of an anaesthetist are not limited to the choice of good anaesthetic agents. He must also pay a close attention to the heart action and to the arterial oxygen saturation. In this the cardiothacoscope and the oximeter may prove to be of great help.

If a cardiac arrest occurs, the anaesthetist should immediately look for close co-operation between the members of the surgical team. Without delay the surgeon performs cardiac massage for adequate circulation. The anaesthetist, on the other hand, administers oxygen under positive pressure. Everyone knows that thoracic surgery often means large amounts of blood lost. This may be immediately estimated by the weighing of the sponges and later on the postoperative ward, by blood volume dye test method. Then the blood volume is maintained at normal level by adequate replacement of blood loss.

The Postoperative Period

For the anaesthetist, the recovery period is as important as the operative one. During the first postoperative hours, the anaesthetist stands by the patient as long as he needs his services. In the following days, in co-operation with the surgeon, the anaesthetist supervises oxygenation, blood transfusion, fluid balance, sedation, aerosol therapy and physiotherapy. The pulse rate serves as a barem for oxygenation; a pulse of one hundred or over per minute is an indication for extending the oxygenation for two hours. The Anaesthesiologic

Department, in our hospital, is also responsible for the Blood Transfusion Service. About one hundred transfusions monthly are given on active surgical wards.

According to fluid balance, the anaesthetist orders fluids and electrolyte solutions.

We routinely try to use less depressive sedatives (Demerol, Levodromoran), infiltration blocks with cyclaine or xylocaine, 5 per cent alcohol solution given intravenously. According to Dr. Mary Knapp of Wesley Memorial, Chicago, 5 per cent alcohol solution given intravenously increases the caloric intake, has a special value for patients with inanition, and reduces by 30 to 50 per cent the need of narcotics postoperatively.

The most serious respiratory complication, atelectasis, is jugulated by carbon dioxide inhalation in association with the physiotherapy practised in the immediate postoperative period; aerosol therapy (Streptomycin, Vaponefrin, Alevaire) and bronchial toilet by aspiration or broncho-aspiration are also helpful. But the use of some anaesthetic techniques has greatly reduced the postoperative respiratory complications for our patients.

As everyone knows, the anaesthetists work more often in the Surgical Department than in the Medical Department; however, our services are also required by latter department to dispense to their patients the same services: oxygenation, blood transfusion, aerosol therapy, nerve blocks, reanimation and also appreciation of anaesthetic risk.

In conclusion, we may say that the recent advances in thoracic anaesthesia have greatly enlarged the thoracic surgical Field.

RÉSUMÉ

Au cours de ce travail, nous allons démontrer le rôle joué par l'anesthésiste dans un centre de chirurgie thoracique en tuberculose.

I. A la période pré-opératoire, l'anesthésiste se doit de bien connaître son patient afin d'être en mesure de le bien préparer à l'intervention. Il doit s'intéresser surtout à l'examen clinique pré-opératoire et aux examens de la fonction cardio-respiratoire. Suivant les renseignements que lui ont fournis ces examens, it doit:

(*a*) restaurer l'équilibre électrolytique et surtout le volume sanguin;
(*b*) instituer de l'aérosolthérapie si nécessaire;
(*c*) diriger le physiothérapeute;
(*d*) décider de la technique anesthésique à employer;
(*e*) ordonner la prémédication la veille de l'opération.

En tuberculose pulmonaire, les problèmes communs à la chirurgie thoracique ne font que s'aggraver.

II. A la période opératoire, une bonne oxygénation est de rigueur, peu importe l'agent anesthésique employé. A l'ouverture de la paroi, l'infiltration intercostale est faite pour prévenir certains réflexes vagaux et la respiration contrôlée ou assistée est pratiquée pour lutter contre la respiration paradoxale et le ballottement médiastinal.

Dans nos milieux, à cause de l'usage courant de l'électro-cautère, nous utilisons

la technique Protoxyde d'azote oxygène avec emploi de substances intra-veineuses : anesthésiques, sédatifs et curarisants. Pour les raisons mentionnées par le docteur Carlens lui-même, dans la revue Anesthesiology de janvier 1953, la sonde Carlens est notre premier choix comme technique d'intubation. A date, elle a été utilisée dans plus de cinq cents cas : résections pulmonaires, décortications, thoracoplasties.

Nous employons aussi le bloqueur Thompson, une sonde endo-bronchique ou une simple sonde endo-trachéale. Dans quelques cas limites au point de vue respiratoire, nous avons aussi employé le "Cocktailytique" de Huguenard et Laborit associé au non à l'anesthésie locale ou régionale.

Au cours de l'opération, le cardiothacoscope et l'oxymètre sont des aides précieux pour l'anesthésiste dans la surveillance de son patient. En présence d'arrêt cardiaque, l'anesthésiste doit pourvoir à l'oxygénation par tous les moyens possibles. Enfin, il doit maintenir le volume sanguin en remplaçant adéquatement le sang perdu.

III. Pour l'anesthésiste, la période post-opératoire est aussi importante que l'opération elle-même. Dans les premières heures de la réanimation, il demeure avec son patient aussi longtemps que l'état de ce dernier l'exige.

Par la suite, de concert avec le chirurgien, il prend charge du service de l'oxygénation, de la transfusion, de l'aérosolthérapie et de la physiothérapie.

En conclusion, nous pouvons dire que les succès actuels de la chirurgie thoracique sont dans une grande mesure dus aux progrès de l'anesthésie thoracique moderne.

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FACTORS AFFECTING TRICHLORETHYLENE VAPOUR CONCENTRATIONS

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This investigation was carried out to determine the trichlorethylene concentrations delivered by a standard anaesthetic apparatus, and to assess the alteration of these concentrations with changes in (*a*) valve opening, (*b*) rate of gaseous flow, (*c*) fluid volume, and (*d*) liquid temperature.

Method

A standard Airmed anaesthetic machine† with a Marrett head was used. The valve openings on the head of the trichlorethylene bottle were graded as 1, 2, 3 and 4 (marked FULL). The trichlorethylene vapour, carried by nitrous oxide, passed through a 36-inch piece of rubber tubing to attain room temperature. A half-litre glass weighing bottle, with valves at its inlet and outlet, could be attached. By experiment, it was found that at a flow rate of five litres or greater, for a period of one minute, complete displacement, for practical purposes, occurred.

The valve opening, volume of trichlorethylene, and the gaseous flow rate were pre-arranged for each experiment. The gas was allowed to flow continuously for 90 minutes. At each fifteen-minute interval, a sample was taken, and the trichlorethylene temperature and volume with the room temperature and atmospheric pressure were recorded.

The sample was collected by connecting the weighing bottle and tubing for one minute. The bottle was then weighed, flushed with nitrous oxide for one minute, and re-weighed. From the change in weight, the trichlorethylene vapour concentration could be calculated using the following formula:

$$\text{Trichlorethylene (vols. \%)} = \frac{22.4}{G} \times \frac{760t}{273p} \times \frac{100}{v} \times \Delta W,$$

where G = the difference in weight at N.T.P. of the gram molecular weights of nitrous oxide and trichlorethylene.
t = ambient temperature in degrees absolute.
p = atmospheric pressure in mm. Mercury.
v = volume of weighing bottle in litres.
W = change in weight of bottle in grams.

Results

The first group of experiments was carried out to determine the effect of altering the gaseous rate of flow. With the initial volume constant (90 cc.), the

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trichlorethylene vapour concentration was determined for each valve opening using flows of 5, 6, 7, 8, 9, and 10 litres per minute.

Using valve opening 4, the vapour concentrations during each experiment varied greatly. The results are presented in Fig. 1 and they also illustrate the high initial concentrations with a rapid fall observed only with valve opening 4. In this group of experiments the gas invariably "bubbled" through the trichlorethylene producing a high degree of vaporization. Isolated examples within these experiments revealed that marked decreases in vapour concentration occurred, despite a constant low temperature.

Using valve openings 1, 2, and 3, "bubbling" was not observed and the vapour concentration, within each experiment, varied only slightly after equilibration. Therefore an average concentration for each whole experiment was taken and is reported in Table I.

TABLE I

TRICHLORETHYLENE VAPOUR CONCENTRATIONS
(vol. %)

L/M	Valve opening 1	2	3
5.	<.1	.1	.8
6.	.1	.4	1.2
7.	.1	.7	.8
8.	.1	.5	.6
9.	.1	.6	.6
10.	<.1	.4	.3

A similar group of experiments was carried out to determine the effect of altering the volume of liquid trichlorethylene. With a constant flow rate of 8 litres per minute, the vapour concentrations were determined for each valve opening, using 30, 60, 90, and 120 cc. of trichlorethylene. These results are reported in Fig. 2 and Table II.

TABLE II

TRICHLORETHYLENE VAPOUR CONCENTRATION
(vol. %)

Volume of liquid	Valve opening 1	2	3
30 cc.	<.1	.9*	1.2*
60 cc.	<.1	.2	.3
90 cc.	.1	.5	.6
120 cc.	<.1	.2	.8*

*Indicates surface disturbance.

A survey of the preceding experiments was made to observe any effect on vapour concentrations due to change in temperature of the trichlorethylene. In half the experiments, there was no detectable change in the trichlorethylene con-

centrations, although a mean fall of 4.2 ± 0.6° C. below room temperature occurred. Where there was a greater fall in temperature, due to bubbling, the average fall below room temperature was 15.4 (± S.D. 0.4)° C. In these experiments it was not possible to separate the reduction in concentration due to cooling from that due to other factors.

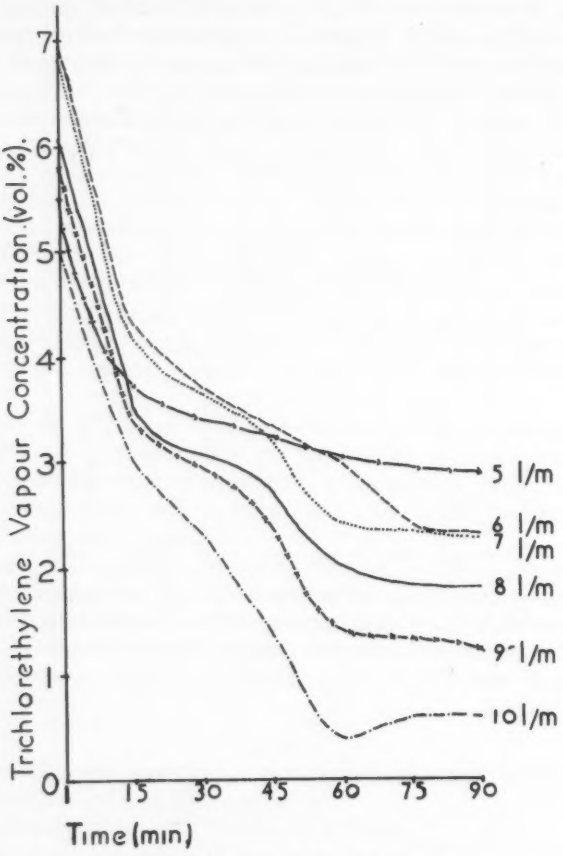

FIG. 1. Valve opening 4; liquid vol. 90 cc.

DISCUSSION

In Fig. 1 is shown the reduction in concentration that occurs with increasing flow rate. When valve opening 4 is used, the rate of vaporization is maximal so that by increasing the flow rate, dilution occurs and the trichlorethylene concentration decreases.

The data shown in Table I were examined by a standard analysis of variance. The difference in vapour concentrations at valve openings 1, 2, and 3 was significant as shown by the f test with $p<0.001$. Similarly it was shown that, in

general, the difference in vapour concentrations at different flows between 5 and 10 litres per minute was not significant ($p>0.05$). However, there would seem to be a tendency with increasing flow rate for the vapour concentrations to reach a maximum and then diminish.

The information in Fig. 2 suggests that a relationship exists between trichlorethylene vapour concentration and liquid volume, when valve opening 4 is used. As the level of liquid is raised, the gas must "bubble" through a greater distance and, consequently, vaporization is increased. When a 30 cc. volume is used, a

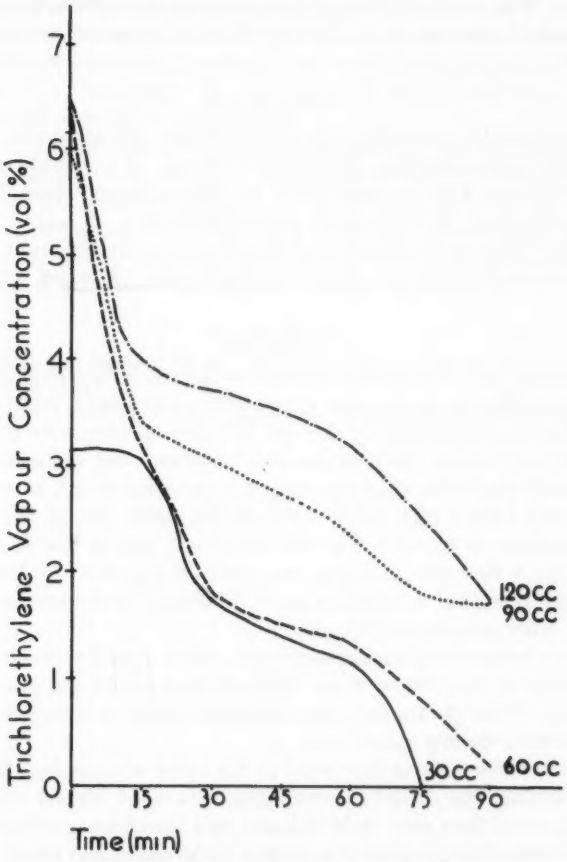

FIG. 2. Valve opening 4; flow rate 8 l/m.

low concentration is observed initially; this is probably owing to the low level of liquid which prevents adequate "bubbling" for maximal vaporization to occur.

The data in Table II indicate that the volume of liquid does not affect the vapour concentrations with valve openings 1, 2, and 3, except where surface dis-

turbance occurs. When the level of trichlorethylene was opposite any of the openings of the central metal core, the surface was agitated and the concentration increased temporarily to a variable degree.

The effect on vapour concentration due to cooling of the trichlorethylene approximately 4.2 degrees below room temperature, was obviously minimal. In experiments with a greater fall in temperature, it was impossible to separate the reduction in concentration due to cooling, from that due to other factors. However, the normal vapour pressure curve of trichlorethylene "flattens" at room temperature (1), so that even a large fall in temperature does not greatly reduce its concentration. The minimal effect of temperature on trichlorethylene concentration is in marked contrast to the known effect of temperature on ether concentrations.

Conclusions

It is evident that the concentrations obtained with valve opening 4 are too high for continuous administration during anaesthesia (2). "Bubbling" should be avoided, since these high concentrations are only obtained when it occurs.

The concentrations obtained with valve openings 1, 2, and 3 lie within the clinical range. They are little affected by alterations in flow rate, fluid volume, and liquid temperature but do show transient minor increases with surface disturbances.

Summary

The trichlorethylene vapour concentrations delivered by a standard anaesthetic machine were determined at room temperature and atmospheric pressure. The effect on these concentrations by changes in valve opening, rate of gaseous flow, fluid volume, and liquid temperature was observed and discussed.

It was shown that with valve opening 4, a progressive fall in vapour concentration occurred from a high initial level. At this valve setting, the concentration tended to decrease as gaseous flow was increased, and to rise as the fluid level was increased. A decrease in liquid temperature appeared to have little effect on vapour concentration, which is in marked contrast to the known effect of temperature on ether concentrations.

The vapour concentrations attained with valve opening 4 are too high for continuous clinical use. Since these high concentrations are always associated with bubbling of the gas through the trichlorethylene, it is suggested that "bubbling" be avoided during anaesthesia.

The vapour concentrations increased as the valve was opened from 1 to 3. At each valve setting, the initial concentration remained almost constant despite changes in gaseous flow rate, fluid volume, and liquid temperature. A slight increase in concentration occurred if a surface disturbance was created. The vapour concentrations attained with valve openings 1, 2, and 3 are within the clinical range.

Résumé

Les concentrations de vapeur de trichloréthylène émises par une machine anesthésique standard ont été déterminées à la température environnante et

sous la pression athmosphérique. On a observé et discuté les effets produits sur ces concentrations par les changements apportés à l'ouverture de la valve, au débit du flot gazeux, au volume du fluide et à la température du liquide.

On a démontré qu'avec une ouverture de valve 4, il se produisait une chute progressive dans la concentration de la vapeur à partir d'une forte proportion initiale. Avec ladite ouverture, la concentration tendait à décroître quand on augmentait le flot gazeux et à s'élever quand on augmentait le débit du fluide. Une baisse dans la température du liquide a paru n'avoir que peu d'effet sur la concentration de la vapeur, ce qui est en net contraste avec les effets connus de la température sur les concentrations d'éther.

Les concentrations de vapeur atteintes avec une ouverture de valve 4 sont trop élevées pour un emploi clinique continu. Ces fortes concentrations étant toujours accompagnées d'un barbotage du gaz dans le trichloréthylène, on suggère d'éviter le barbotage pendant l'anesthésie.

Les concentrations de vapeur augmentaient quand on ouvrait la valve de 1 à 3. Pour chaque position de la valve, la concentration initiale restait presque constante malgré les changements apportés au débit gazeux, au volume du fluide et à la température du liquide. Il se produisait une légère élévation de concentration si l'on en agitait la surface. Les concentrations de vapeur atteintes avec des ouvertures de valve 1, 2 et 3, sont du domaine clinique normal.

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ANAESTHETIC DOSAGE IN THIOPENTAL–CURARE–NITROUS OXIDE ANAESTHESIA, USING A THIOPENTAL–CURARE MIXTURE

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THE advance presented by the introduction of short-acting barbiturates and muscle relaxants in anaesthesiology is recognized universally. However, the very ease with which patients are anaesthetized by this technique makes it more exacting for the anaesthetist than earlier methods.

Following unfortunate experiences with barbiturates and muscle relaxants in their early usage it was stated repeatedly that these drugs were dangerous when used in combination for lengthy procedures (1, 2). Another school of thought practised the restriction of barbiturates to the induction period, after which the patients were switched to some other anaesthetic for maintenance (3). Additional criticism was directed against the use of a thiopental–curare mixture (Baird's solution) (4) as it was thought to reduce the flexibility of the technique.

Contrary to these sentiments our impression is that properly managed barbiturate–curare–N_2O anaesthesia is among the better and safer anaesthetic techniques available today; and its administration is further facilitated by adoption of the thiopental–curare mixture in lieu of the separate agents.

In view of this divergence of opinion it appeared desirable to examine more closely the time-dosage relationship in thiopental–curare–N_2O anaesthesia and to compare it with data from the literature regarding the separate use of the same agents. It was hoped that the results would permit conclusions about the relative merits of the two techniques.

MATERIAL AND METHODS

This investigation comprises 86 cases lasting from 1 to 9 hours. One anaesthesiologist managed 71 cases; the remaining 15 were conducted by several others. The anaesthetic experience of these individuals varied considerably. In each case the time and amount of every dose of thiopental–curare was noted on the anaesthetic chart. A fixed concentration of 50 per cent nitrous oxide and 50 per cent oxygen was inhaled by all patients throughout the operations. The types of procedure, age of the patients, and anaesthetic utilization are presented in Table I.

The rate of anaesthetic utilization was determined for each patient according to the following formula:

$$R = \frac{\text{thiopental–curare mixture used (mg/kg)}}{\text{anaesthesia time}}$$

These rates were grouped into six classes according to length of anaesthesia. The average rates of utilization for each class were calculated. The standard error of the means was determined by the accepted formula (5):

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$$SE_M = \sqrt{\frac{N\sum x^2 - (\sum x)^2}{N^2(N-1)}}$$

The average totals of anaesthetic used in each class were determined by multiplying the average utilization rates by the midpoint anaesthesia times. The average intubation dose also was determined along with the average time that had elapsed from the administration of the intubation dosage to the administration of the next increment of thiopental–curare mixture. Although 78 patients in this series were intubated, data from only 49 cases were available for the latter determination.

TABLE I

Type of operation	1–2	2–3	3–4	4–5	5–6	6+	TOTAL
	HOURS ANAESTHESIA TIME						
Head and neck	2	3	4	—	—	—	9
Open chest	2	4	8	8	3	4	29
Intraperitoneal	3	5	9	9	4	3	33
Others	4	8	2	1	—	—	15
Age groups							
–2	1	—	1	—	—	—	2
2–15	2	2	2	2	1	—	9
15–60	8	12	15	11	3	6	55
60+	—	6	5	5	3	1	20
*Thiopental-curare mixture used (cc)**							
–20	4	1	1	2	—	—	8
20–40	6	9	11	7	—	1	34
40–60	1	10	8	8	6	2	35
60+	—	—	3	1	1	4	9
TOTAL	11	20	23	18	7	7	86

*Each cc of thiopental-curare mixture contains 25 mg. thiopental and 0.75 mg. d-tubocurare.

Results

A representative example of individual time-dose curves is shown in Fig. 1.

The average utilization rates are plotted against the midpoints of the anaesthetic time classes in Fig. 2.

The averages of total utilization are plotted against the midpoints of the anaesthetic time classes in Fig. 3. An additional point is gained in this coordinate system by plotting the average intubation dosage against the average time after which the next increment became necessary.

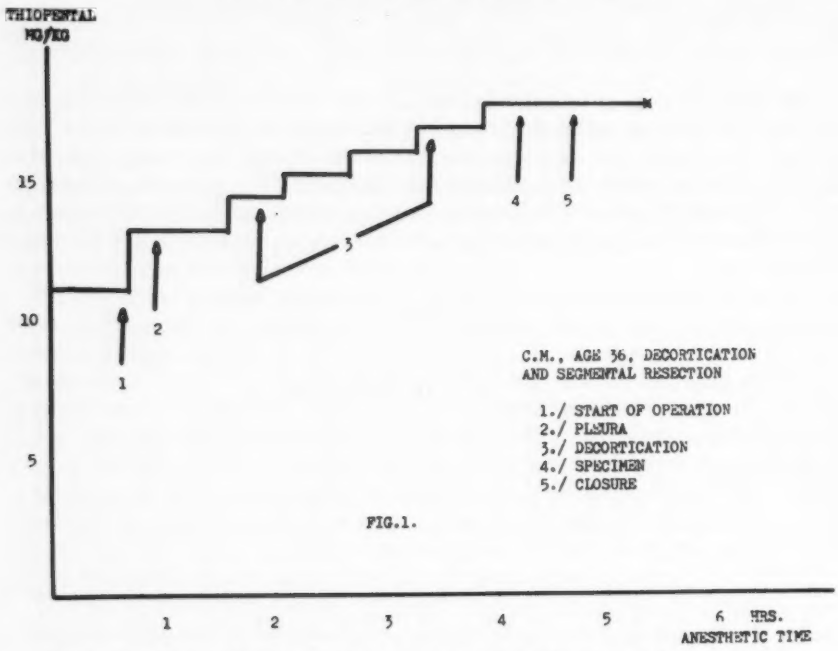

FIG. 1. Sample cumulative time-dose curve. Patient intubated at 0 hr., awake and transfered to recovery room at *X*.

DISCUSSION

The most surprising feature revealed by the individual time-dose curves was that sudden variations in the degree of surgical stimulus, such as occur in the course of every operation, were not accompanied by large enough changes in drug requirements to distort the curvilinear relationship. Theoretically such behaviour would be explicable if the patients were carried at too deep a plane of anaesthesia all the time. By all clinical criteria available this was not the case.

The smoothness of the curve shown in Fig. 2 appears all the more remarkable because the number of observations is relatively small and the data were obtained under operating room conditions by several anaesthesiologists working with different surgical teams.

Fig. 3 represents the average dosage of thiopental–curare mixture in the course of an operation. This claim needs further justification, because all but one of the points on which the curve is based were derived from average total anaesthetic utilizations of individual cases. It might be argued that though the endpoints may fall along a curve, it does not follow that the individual curves themselves resemble the curve thus defined. The belief that little error if any was introduced through this assumption is based on inspection of the 86 individual time-dose curves. They all appear similar to Fig. 3 and to one another. It is strengthened

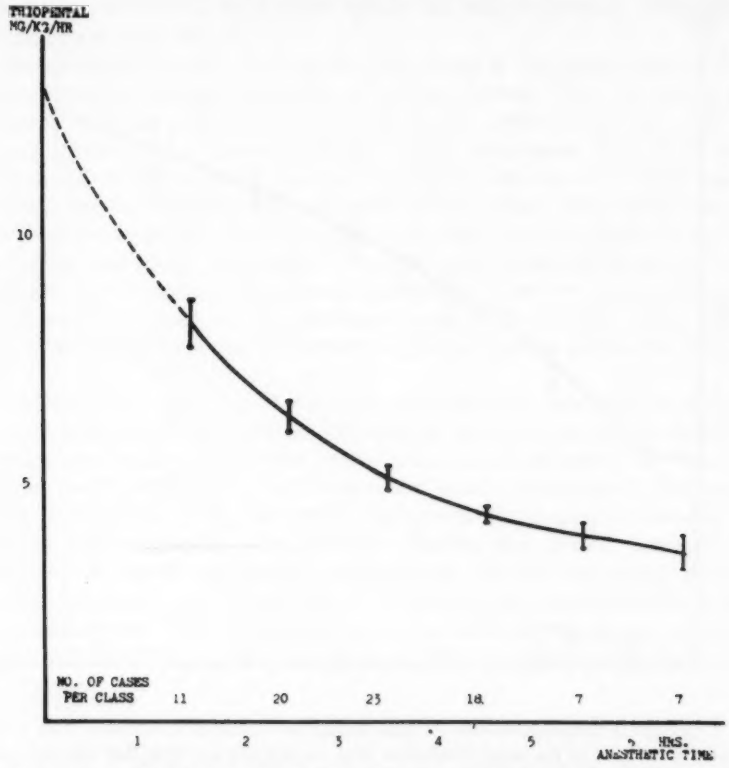

FIG. 2. Average utilization rates of thiopental–curare mixture, grouped into six classes according to the duration of anaesthesia. The vertical lines indicate the standard error of means.

further by the good fit of the point computed from the intubation dosages to the curve that is otherwise defined by total dosages.

It follows from the way in which the curve in Fig. 3 was constructed that it will indicate the cumulative dosages required to maintain, for a given time, that clinical level of anaesthesia which is prevalent during the major part of the procedure. The difference between the (extrapolated) induction dose and the actual intubation dose is explained by the observation that intubation ordinarily requires a deeper initial level of anaesthesia than induction without intubation.

The maintenance of a clear airway and adequate respiratory exchange are among the principal safeguards of thiopental–curare–N_2O anaesthesia. These objectives are facilitated best by intubation. The widespread reluctance to perform intubation has been based occasionally on the fear that it will "unnecessarily" increase the dosage of anaesthetics. Fig. 3 objectifies the clinical impression that in procedures lasting over 45 minutes intubation will NOT increase the total anaesthetic consumption in the average patient.

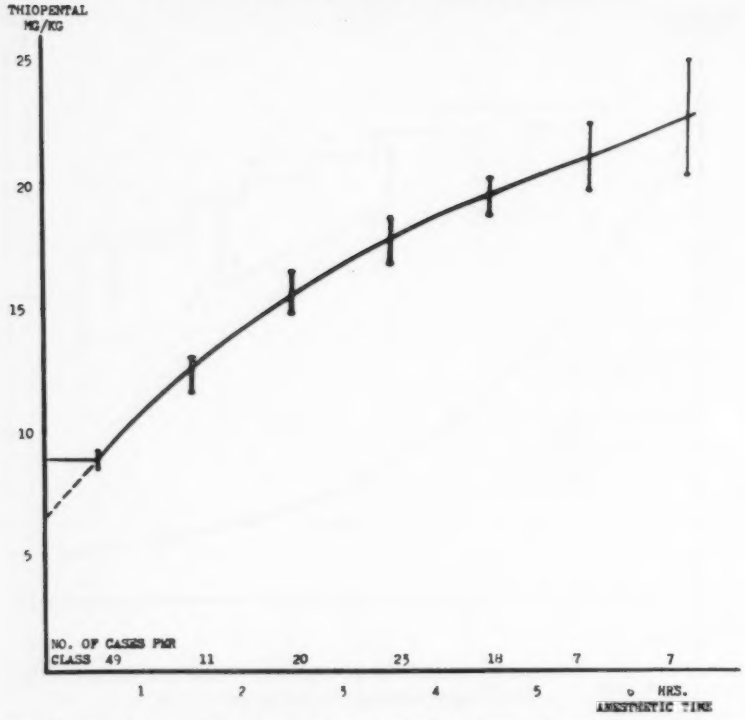

FIG. 3. Dosage of thiopental–curare mixture during the course of anaesthesia. The first class (49 cases) is the plot of the mean intubation dose vs. the average time that elapsed between its administration and the administration of the next increment. All other classes represent average total utilizations of thiopental–curare plotted against the class midpoint anaesthesia times. Vertical lines indicate standard errors of the means.

In all procedures respirations were vigorously assisted throughout. The assistance was of the same order that proved adequate in other investigations (6) to maintain normal or lower than normal alveolar CO_2-levels. The effectiveness of relatively small amounts of thiopental–curare mixture probably was due primarily to this factor (7).

Using a similar anaesthetic technique but separate syringes of thiopental and curare, Dundee (7) arrived at 8.5 mg. thiopental per kg. body weight as the induction dose and 12.46 mg/kg as the 90-minute value. Our corresponding data were 8.6 and 12.3 mg/kg respectively.* The thiopental–curare ratios in Dundee's work were 31:1, 24:1, 29:1, and 16:1 at different points in the course of anaesthesia with varying degree of respiratory assistance. The thiopental–curare ratio in our mixture is 33:1. It thus appears that with thiopental–curare mixture satisfactory operative conditions can be achieved by using probably less but certainly

*In an article published since the completion of this study (Canad. Anaes. Soc. J. 2:41, 1955) C. F. Egan finds the average intubation dose of thiopental for infants 9 mg/kg with the use of Baird's solution. In view of the fact that weight for weight infants require more anaesthetic than adults this finding is in excellent agreement with the present data.

not more curare than when the two agents are used separately. The thiopental consumption is about equal.

It should be emphasized that the surgical teams in this series insisted on profound, practically apnoeic relaxation of all the patients. Even so it was a rare occurrence that the patients were not sufficiently awake at the end of the procedure to maintain their airway and obey simple commands. Some of those who were not responding could be induced to respond after the administration of an anticurare agent, indicating that on these occasions they were overcurarized to some degree even at this ratio. Comparison of data from two institutions, as was done in the preceding paragraph, is fraught with uncertainties because of the differences in an unknown number of conditions. However, the validity of the conclusions drawn here can be challenged only in the unlikely event that it is shown that Dundee's series consistently required greater relaxation than these cases.

As indicated in Figs. 1 and 3, the bulk of the mixture was given at a fast rate during induction. If the patient happens to react adversely to one of the pharmacologic agents, this is the period when accidents would be most likely to occur, provided the rest of the anaesthesia is not mismanaged. Following induction the balance of the thiopental–curare mixture is given in smaller increments at ever increasing time-intervals. During this period there is ample opportunity to detect and correct complications. Should they occur, alternative agents and methods may be considered. Otherwise the safest practice is to continue maintenance with thiopental–curare, for there seems to be no reason to consider the length of a procedure *per se* to be a contra-indication to the use of these agents.

Summary

1. Analysis of 86 cases in which a thiopental–curare mixture (Baird's solution) was used, and a comparison with data obtained by others who administered the drugs separately, indicate that use of the mixture does not result in an increase of the dosage of either agent.

2. Time-dose curves are presented to show that a definite curvilinear relationship exists between drug-utilization and the duration of anaesthesia.

3. In cases lasting over 45 minutes intubation will not increase the anaesthetic consumption.

Résumé

On a déterminé les besoins anesthésiques de 86 malades soumis à l'anesthésie au thiopental-curare (la solution de Baird). On a découvert qu'il existait une relation curviligne très nette entre l'utilisation de la drogue et le temps d'anesthésie. La comparaison des présents énoncés avec ceux d'autres chercheurs qui ont administré le thiopental et le curare séparément, mais selon la même technique, indique qu'on a conservé le flexibilité de la technique en employant le mélange standard et qu'on pourrait réaliser une certaine économie de curare. Les résultats montrent aussi que, dans les interventions d'une durée supérieure à 45 minutes, l'intubation n'accroît pas les besoins anesthésiques totaux du malade moyen.

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A COMPARISON OF THE ANTI-EMETIC EFFECTS OF DIMENHYDRINATE, PROMETHAZINE HYDROCHLORIDE AND CHLORPROMAZINE FOLLOWING ANAESTHESIA

DOREEN CAPLIN, M.D.*, and CODE SMITH, M.D.**

VOMITING during the immediate postoperative period is a frequent complication of anaesthesia in children. Dimenhydrinate, promethazine hydrochloride, and chlorpromazine have all been recommended as effective anti-emetic drugs (1, 2, 3). A comparison of the clinical efficacy of these three agents was made with a carefully selected and controlled group of patients.

A group of patients undergoing operation for correction of squint was chosen for this study for the following reasons:

1. They were reputed to have a relatively high incidence of post-anaesthetic vomiting, a prerequisite for the demonstration of anti-emetic action in a relatively small series of cases.
2. There was no operative interference with or disease of the gastro-intestinal tract.
3. All operations lasted between one-half and one hour.
4. All patients were on the same ward with the result that observations were made by the same nursing personnel throughout the project.
5. All cases were elective and had received nothing by mouth for at least four hours prior to induction, and had received no solid food since the evening prior to operation.

The cases used in the study were drawn from both public and private patients of certain eye-surgeons and were either treated or placed in the control group on a completely random basis.

The first comparison was made between 40 cases treated with dimenhydrinate and 40 untreated controls. Towards the completion of this series, optimistic reports of the effectiveness of promethazine hydrochloride and chlorpromazine prompted us to conduct a second series consisting of 20 cases treated with promethazine hydrochloride, 25 cases treated with chlorpromazine and 20 untreated controls.

ANAESTHETIC MANAGEMENT

All cases were treated in the following routine manner:

1. All had atropine alone for preoperative medication, and narcotic drugs, alleged to contribute to post-anaesthetic vomiting, were not administered.
2. Peroral endotracheal intubation was performed in all cases following induction with pentothal sodium and relaxant whenever venepuncture was feasible, or, in smaller children, with ethyl chloride–ether sequence.

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**Hospital for Sick Children, Toronto.

3. Maintenance consisted of nitrous oxide, oxygen, trichlorethylene and/or ether. Three groups can thus be defined:

(i) patients receiving ethyl chloride and ether,

(ii) patients receiving pentothal sodium, nitrous oxide, oxygen, relaxant, and ether,

(iii) patients receiving pentothal sodium, nitrous oxide, oxygen, relaxant, and trichlorethylene.

4. After the child was intubated, the selected drug was administered by the intramuscular route. Dosages employed were:

Dimenhydrinate–1 mg./lb.
Promethazine hydrochloride –1 mg./3 lb.
Chlorpromazine–1 mg./3 lb.

Postoperative Handling

The recovery room nurses and the nurses on the ward were informed of the project and their co-operation was obtained. They were not told which of the children had received the drug. On every chart a note was placed to remind the nursing staff to record whether or not the patient vomited, the time and the approximate amount, and the volume of fluid intake.

Control Series

Sixty untreated controls were studied. Of these, 39 were given ethyl chloride and ether; 21 had pentothal solium, relaxant, notrous oxide, and oxygen. Ether was added to the mixture in 11 of the latter; trichlorethylene was added in the remaining 10 cases. The presence or absence of vomiting as well as the average number of emeses (when present) are shown in Table I.

TABLE I

Agents	Number of cases	Vomiting	No vomiting	No. of emeses/case	No. of emeses/vomiter
Ethyl chloride and ether	39	28	11	2.1	2.9
Pentothal sod. and ether	11	9	2	2.7	3.3
Pentothal sod. and trilene	10	4	6	0.8	2.0

When ether was administered, the number of emeses remained approximately the same, irrespective of the type of induction. However, there was some improvement when ether was omitted from the sequence.

Results of Treatment

(Throughout the analysis, P values were obtained by the application of the method of chi square (X^2) to the enumeration data and by the calculation of t values for the mensuration data.)

The over-all incidence of vomiting for treated and control groups is shown in Table II. Treatment resulted in a significant decrease in the incidence of post-operative vomiting. ($P < 0.05$).

TABLE II

Treatment	Number of cases	Vomiting	No vomiting	No. of emeses/case	No. of emeses/vomiter
Control	60	41	19	2.0	3.0
Treated	85	41	44	1.0	2.0
Dimenhydrinate	40	18	22	1.0	2.2
Chlorpromazine	25	9	16	0.6	1.7
Promethazine hydrochloride	20	14	6	1.4	1.9

The administration of chlorpromazine reduced the incidence of vomiting to a significant degree ($P < 0.02$). There was no statistically significant reduction in the incidence of vomiting in the series treated with dimenhydrinate or promethazine hydrochloride. However, in the group that did vomit, the frequency of vomiting was reduced by all drugs.

1. Group (i) Anti-Emetic Drugs with Ethyl Chloride, Ether Anaesthesia.

The results of this group are given in Table III.

TABLE III

Treatment	Number of cases	Vomiting	No vomiting	No. of emeses/case	No. of emeses/vomiter
Control	39	28	11	2.1	2.9
Dimenhydrinate	24	15	9	1.5	2.4
Chlorpromazine	15	6	9	0.7	1.8
Promethazine hydrochloride	15	10	5	1.5	2.3

The incidence of vomiting was reduced to a significant degree by chlorpromazine only ($P < 0.05$).

2. Group (ii) Anti-Emetic Drugs with Pentothal Sodium, Nitrous Oxide, Oxygen, Relaxant, and Ether Anaesthesia

Because of the small number of cases, the treated groups were combined for the purposes of comparison in Tables IV and V.

TABLE IV

Treatment	Number of cases	Vomiting	No vomiting	No. of emeses/case	No. of emeses/vomiter
Control	11	9	2	2.7	3.3
Treated	21	6	15	0.3	1.0

TABLE V

Treatment	Number of cases	No vomiting	Vomiting	No. of emeses/case	No. of emeses/vomiter
Control	10	4	6	0.8	2.0
Treated	14	4	10	0.5	1.3

In this group, treatment resulted in a significant decrease in vomiting. ($P < 0.02$).

3. Group (iii) Anti-Emetic Drugs with Pentothal Sodium, Nitrous Oxide, Oxygen, Relaxant, and Trichlorethylene Anaesthesia

The treated groups were again combined. As noted in Table I, the incidence of vomiting was low in the untreated group and no improvement was observed on administering anti-emetic drugs.

We considered that a favourable anti-emetic effect of a single injection of any drug should consist of

(1) a reduction in frequency of postoperative vomiting and/or

(2) a delay in the onset of vomiting, if it occurred.

An average number of emeses/case together with an average delay in onset of vomiting in hours is shown in Table VI. For purposes of calculation, those patients which did not vomit were considered to have a delay in vomiting equivalent to ten hours. Table VI also shows an arbitrary rating applied to all groups. To obtain this rating, a point value was assigned to each postoperative response on the basis of the two criteria listed above, and an average calculated.

No vomiting postoperatively	5 points
Single emesis 3 hours or more postoperatively	4 points
Repeated emeses starting after 3 hours postoperatively	3 points
Single emesis within the first 3 hours postoperatively	2 points
Repeated emeses starting within the first 3 hours postoperatively	1 point

TABLE VI

Treatment	Onset of vomiting (hours)	Emeses/case	Rating
Control	4.2	2.0	2.8
Dimenhydrinate	6.1	1.0	3.6
Chlorpromazine	7.6	0.6	4.3
Promethazine hydrochloride	4.6	1.4	3.2

According to this scale, chlorpromazine exhibits a highly significant increase in rating over the control value and dimenhydrinate exhibits a significant increase in rating. Promethazine hydrochloride was without effect.

Disadvantages

The only disadvantage of treatment noted during the study was a sedative effect which could lead to a reduction in fluid intake. Accordingly, the sedative effect was studied by observing the average time to regain consciousness. ("Conscious time" is defined as the time from admission to the recovery room until the child answers questions coherently.)

Each anti-emetic drug increased the period of unconsciousness postoperatively as shown in Table VII.

TABLE VII

	Control	Dimenhydrinate	Chlorpromazine	Promethazine hydrochloride
Aver. conscious time (minutes)	31	41	37	49

The fluid intake was studied by noting volumes of liquid ingested and vomited by each child until midnight of the day of operation. The entire series was divided into 2 classes—vomiters and non-vomiters—and average net fluid intakes were calculated for both classes in each of the four series. (See Table VIII.)

Each anti-emetic drug reduced the average fluid intake but only to a slight degree for both classes. When a combined average net intake (vomiters and non-vomiters) was calculated for each series, promethazine hydrochloride was found to be the only drug which decreased the fluid intake.

Summary and Conclusions

One hundred and forty-five patients undergoing operation for the correction of squint were studied with respect to the effectiveness of three anti-emetic

TABLE VIII

Treatment	Non-vomiters aver. fluid intake (ounces)	Vomiters			Combined aver. net fluid intake (ounces)
		Aver. fluid intake (ounces)	Aver. output (ounces)	Net (ounces)	
Control	18	14	6	8	11
Dimenhydrinate	17½	13	2½	10½	14
Chlorpromazine	14	13	3½	9½	12
Promethazine hydrochloride	17	12	5½	6½	10

drugs: Dimenhydrinate (40 cases), Promethazine hydrochloride (20 cases), Chlorpromazine (25 cases), and a control group (60 cases).

Chlorpromazine reduced the incidence of vomiters to a significant degree, reduced the frequency of vomiting in cases that did vomit, produced only a slight prolongation in the time of recovery from anaesthesia, and did not decrease the average fluid intake.

Dimenhydrinate reduced the incidence of vomiters and frequency of vomiting to a less marked degree than chlorpromazine, produced a somewhat more prolonged recovery time than chlorpromazine, and did not decrease fluid intake.

Promethazine hydrochloride did not significantly decrease the incidence of cases of vomiting post-operatively, although the frequency of vomiting was decreased. Recovery time was more prolonged than with either of the other drugs and the fluid intake was slightly reduced.

We conclude that, of the three drugs investigated, chlorpromazine is the most effective anti-emetic drug with a minimum of side-effects in children undergoing operation for the correction of squint.

Acknowledgments

Acknowledgments are made to the Staff in Ophthalmology and Anaesthesia, Hospital for Sick Children, for their co-operation in making this study possible, and to the nursing personnel of the Recovery Room and the Eye Ward for their assistance in maintaining records.

We also wish to thank Frank W. Horner & Co. for the supply of dimenhydrinate (Gravol) and Poulenc Ltd. for the supply of chlorpromazine (Largactil) and promethazine hydrochloride (Phenergan).

Résumé

On a étudié cent quarante-cinq malades soumis à une opération pour la correction du strabisme sous le rapport de l'efficacité de trois médications antiémétiques: Dimenhydrinate (40 cas), Chlorhydrate de Prométhazine (20 cas), Chlorpromazine (25 cas); il y avait un groupe-témoin (60 cas).

La *Chlorpromazine* a réduit l'incidence des vomissements dans une appréciable proportion, a réduit la fréquence des vomissements dans les cas où ils se produisaient, a produit seulement une légère prolongation de la période de rétablissement post-anesthésique et n'a pas diminué l'absorption moyenne de fluide.

Le *Dimenhydrinate* a réduit l'incidence et la fréquence des vomissements à un degré moindre que la chlorpromazine, a provoqué un allongement sensible de la période de rétablissement et n'a pas diminué l'absorption de fluids.

Le *Chlorhydrate de Prométhazine* n'a pas sensiblement diminué l'incidence des vomissements post-opératoires, bien que la fréquence en ait été diminuée. La période de rétablissement a été prolongée davantage qu'avec les deux autres drogues et l'absorption de fluids a été légèrement réduite.

On conclut que, des trois drogues étudiées, la Chloropromazine est le plus efficace anti-émétique présentant un minimum d'effets secondaires chez les enfants soumis à une opération pour la correction du strabisme.

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CANADIAN ANAESTHETISTS' SOCIETY

ANNUAL MEETING

1955

Programme—Monday, June 20

8:30 a.m.—Registration

9:30 a.m.—"Newer Drugs as Preoperative and Postoperative Medication"
ANDRÉ JACQUES, M.D., F.R.C.P.(C).

10:00 a.m.—"The Design of Anaesthetic Inhalers"
SIR ROBERT MACINTOSH, M.D., F.R.C.S., F.F.A.R.C.S., D.A.

10:30 a.m.—"Hypothermia for Cerebral Vascular Surgery"
S. L. VANDEWATER, M.D., F.R.C.P.(C).

11:00 a.m.—"Untoward Reactions to Succinyl-Choline"
JOHN I. DAVIES, M.D., F.R.C.P.(C).

11:30 a.m.—"The Relation of Plasma Cholinesterases to Response to Clinical Doses of Succinyl-Choline"
WERNER KALOW, M.D.

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LUNCHEON

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2:00 p.m.—"Chlorpromazine and Promethazine in Obstetrics"
J. J. CARROLL, M.D.

2:30 p.m.—"Barbiturates in Obstetrics: Effects on the Newborn"
ROLAND DUCHESNE, M.D.

3:00 p.m.—"The Place of Pentothal in Modern Anaesthesia"
RONALD JARMAN, D.S.C., L.R.C.P.(Lond), M.R.C.S.(Eng), F.F.A.R.C.S., D.A.

3:30 p.m.—"Nalorphine, an Opiate Antagonist"
F. PRESCOTT, M.D., M.SC., M.R.C.P.

4:30 p.m.—Meeting of the Council, 1954–55.

Programme—Tuesday, June 21

9:00 a.m.—"Evaluation of Methods of Resuscitation"
GUY FORTIN, M.D.

9:30 a.m.—"Reflexions on the Muscle Relaxants"
GEOFFREY ORGANE, M.A., M.D., F.F.A.R.C.S.

10:00 a.m.—Round Table Discussion: "What is the Place of Induced Hypotension in Anaesthesia?"
Chairman: WESLEY BOURNE, M.SC., M.D., C.M., F.R.C.P.(C), F.F.A.R.C.S.

11:00 a.m.—Annual General Meeting.

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LUNCHEON

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2:30 p.m.—"Considerations of Carbon Dioxide in Anaesthesia"
W. G. CULLEN, M.D.

3:00 p.m.—"Ketosis in Anaesthesia"
A. R. HUNTER, M.D., F.R.F.P.S.G., F.F.A.R.C.S.

3:30 p.m.—"Twenty Years of Anaesthesia for Thoracic Surgery"
GEORGES COUSINEAU, M.D., F.F.A.R.C.S., L. LEGAULT, M.D.
and LEO FOREST, M.D.

4.00 p.m.—"Hibernation: Medical and Surgical Possibilities"
GÉRARD MIGNEAULT, M.D.

4:30 p.m.—Meeting of Council, 1955–56.

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7.00 p.m.—Annual Dinner. Speakers—SIR ROBERT MACINTOSH
WESLEY BOURNE.

Programme—Wednesday, June 22

9:00 a.m.—Round Table Discussion: "Anaesthesia and the Open Chest"
Chairman: S. M. CAMPBELL, M.D., F.R.C.P.(C), F.F.A.R.C.S., D.A.

10:00 a.m.—"Contamination with Phenol"
R. W. COPE, L.R.C.P., M.R.C.S., F.F.A.R.C.S., D.A.

10:30 a.m.—"Anaesthesia for Cardiac Surgery in Children"
CODE SMITH, M.D.

11:00 a.m.—"Anaesthetic Deaths"
E. A. GAIN, M.D.

11:30 a.m.—"Myotonia Congenita: A Complicating Factor in Anaesthesia"
MARIUS DUBEAU, M.D.

B.M.A. & C.M.A.: SECTION OF ANAESTHESIA

Programme, 1955

Wednesday, June 22

2:00 p.m.—"Physiology and Complication of the Trendelenburg Position"
C. LANGTON HEWER, M.B., M.R.C.P., F.F.A.R.C.S.

2:30 p.m.—"Whither Now in Anaesthesia?"
H. R. GRIFFITH, M.M., B.A., M.D., C.M., F.R.C.P.(C), F.F.A.C.S., D.A.

3:00 p.m.—"Antidotes to Curarizing Drugs"
A. R. HUNTER, M.D., F.R.F.P.S.G., F.F.A.R.C.S.

3:30 p.m.—"A New Technique of Intubation for the Prevention of Contralateral Infection in Pulmonary Surgery"
LÉON LONGTIN, B.A., M.D., F.R.C.P.(C).

4:00 p.m.—"Anaesthesia for Children and the Newborn"
R. W. COPE, L.R.C.P., M.R.C.S., D.A., F.F.A.R.C.S.

4.30 p.m.—"Spirometry in Assessment of Analgesia after Abdominal Surgery"
PHILIP R. BROMAGE, M.E., B.S., F.F.A.R.C.S., D.A.

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Thursday, June 23

2:00 p.m.—"Chlorpromazine"
D. A. BUXTON HOPKIN, M.D., F.F.A.R.C.S.

2:30 p.m.—"History of Anaesthesia in Canada"
H. J. SHIELDS, B.A., M.B., F.R.C.P.(C), F.F.A.R.C.S., D.A.

3:00 p.m.—"The Anaesthetic and the Anaesthetist"
RONALD JARMAN, D.S.C., L.R.C.P.(Lond), M.R.C.S.(Eng), F.F.A.R.C.S., D.A.

3:30 p.m.—"Considerations on Recovery Rooms"
FERNANDO HUDON, M.D., F.R.C.P.(C), F.F.A.R.C.S.

4:00 p.m.—Round Table Discussion: Questions to be submitted from the Meeting.
Chairman: B. C. LEECH, O.B.E., M.D., C.M., F.F.A.R.C.S.

MEETINGS

CANADIAN ANAESTHETISTS' SOCIETY, WESTERN REGIONAL MEETING, Regina, Saskatchewan—April 21, 22, 23, 1955

Correspondence should be addressed to
Dr. J. E. McCutcheon,
P.O. Box 34, Regina, Sask.

CANADIAN ANAESTHETISTS' SOCIETY, ANNUAL MEETING, Toronto—June 20, 21, 22, 1955

Programme in this Journal.

BRITISH MEDICAL ASSOCIATION and CANADIAN MEDICAL ASSOCIATION, SECTION OF ANAESTHESIA, Toronto—June 23, 24, 1955

Programme in this Journal.

WORLD CONGRESS OF ANAESTHESIOLOGISTS, Scheveningen, Holland—September 5–10, 1955

Correspondence should be addressed to Mr. W. A. Fentener van Vlissingen, Executive Manager, World Congress of Anaesthesiologists, Bilthoven, Holland. The congress fee will be 35 Dutch Guilders.

NEWS LETTER

The following candidates who attended the postgraduate course in Anaesthesia in the Department of Anaesthesia of the Hôtel-Dieu de Québec, under the direction of Dr. Fernando Hudon, F.R.C.P.(C), F.F.A.R.C.S., were successful in passing the examinations for certification by the Royal College of Physicians and Surgeons of Canada in 1954, and have taken up the appointments designated:

Dr. François Cliche—anaesthetist at Hôpital Notre-Dame, St. Georges, Beauce County.
Dr. Henri Durand—anaesthetist at Hôpital St. Sacrement, Quebec City.
Dr. Patrick Laughrea—anaesthetist at Hôpital St. Joseph, Thetford Mines, P.Q.
Dr. Jacques Fournier—anaesthetist at Alma Hospital, Almaville, P.Q.
Dr. Jacques Houde—anaesthetist in Department of Anaesthesia, Hôtel-Dieu de Québec.
Dr. R. Cruger—anaesthetist at Sea View Hospital, Ottawa.

Major Earl Russell, R.C.A.M.C., has recently been demobilized and has been appointed resident anaesthetist at the Kingston General Hospital.

Dr. Charles Egan, M.B.E., was successful in the recent examinations for certification in Anaesthesia. Dr. Egan has recently been appointed as chief anaesthetist at the Women's Hospital, in Philadelphia.

Dr. V. A. Nekus, Kingston; Dr. Harold Cameron, St. Catherines; Dr. Arthur Sheridan, Montreal; Dr. Charles Egan, Philadelphia; and Dr. Ruth Titcombe (née Ruth Whitfield), Winnipeg, are all former members of the resident staff in Anaesthesia, at the Hôtel-Dieu Hospital, Kingston, who were successful in the certification examinations in 1954. Another member of the group, Dr. Robert Ferguson, Montreal, received his diploma in Anaesthesia, at McGill University, in June.

Dr. W. A. Campbell, Head of the Department of Anaesthesia at Queen's University, is also busily engaged as President of the Canadian Rugby Union.

Major Homer Edwards, R.C.A.M.C., has recently returned from Korea and has been posted as anaesthetist at Kingston Military Hospital.

Many members will remember with pleasure meeting Dr. Lucien Morris at the Annual Meeting at the Seigniory Club, in 1953. Dr. Morris has recently been appointed Professor of Anaesthesiology at the University of Washington.

Our members will read with interest the recent articles emanating from the Department of Anaesthesiology at Duke University, under the direction of Professor Ronald Stephens.

The Western Canadian Divisions will meet in Regina on April 21, 22, and 23. Dr. J. E. McCutcheon is Chairman of the Programme Committee.

Dr. Harold T. Kay, formerly of London, England, who was resident anaesthetist at the Ottawa General Hospital from July, 1953, until July, 1954, has recently been appointed to the active staff in Anaesthesia.

Dr. Maurice Mainville, a member of the staff in Anaesthesia of the Ottawa General Hospital, recently undertook a special assignment for the Department of Immigration and spent an enjoyable year in England and on the Continent.

The Department of Anaesthesia at the Ottawa General Hospital has recently been delegated as a sub-department of Surgery (Anaesthesia) of the Faculty of Medicine, University of Ottawa. Dr. Douglas Law heads the Department, with the rank of Assistant Professor.

Dr. Donald Whyte has been promoted to the rank of Senior Anaesthetist at the Ottawa Civic Hospital. Dr. John Green was recently appointed to the staff.

Drs. Bonfield, Durocher, Kay, Law, and Mainville, of the Ottawa General Hospital staff, recently opened a central business office and report that the move has been completely satisfactory.

Dr. John Cameron has recently entered the private practice of Anaesthesia in Sudbury, Ontario.

In the Sudbury hospitals, a recent innovation has been the provision of a police whistle for each operating room. In case of cardiac arrest, the blowing of this whistle is the signal for the prompt establishment of the necessary emergency measures.

Dr. Orange has on record a case of venticular fibrillation with complete re-establishment of cardiac function within two minutes. The patient made a complete recovery and was discharged from hospital within ten days. Dr. Orange will be glad to provide a complete record of this case to anyone interested in collecting records of these cases.

Dr. Eric H. Dobbs and Donald F. Cameron, both of Edmonton, were successful in the recent examinations for certification in Anaesthesia.

Dr. Donald Kyle has joined the Department of Anaesthesia of the University Hospital, in Edmonton, after completing a period of training at the Vancouver General Hospital.

Dr. Christin Mason (née Howse) is now practising in Calgary.

Dr. John E. Fox of the Royal Alexandra Hospital staff, in Edmonton, is spending a year of postgraduate training in Montreal.

Dr. Norman E. Foster, of Calgary, is spending a year at the University of Alberta Hospital, in Edmonton.

Doctor Norman Urie, a final-year diploma course student at McGill University, has recently returned from the United Kingdom, after having spent a very profitable six-month period visiting various British centres on a Wyeth Fellow-

ship. Doctor Urie has been appointed to the Resident staff of St. Mary's Hospital, Montreal, with duties to commence February 1.

Léon Longtin, M.D., F.R.C.P., Professor of Anaesthesia at the University of Montreal, has been for the past three months acting in an instructional capacity as delegate in Anaesthesia at the Franco-Canadian Institute in Paris, France.

The Windsor group of anaesthetists held their monthly meeting at the Grace Hospital on January 28. Dr. Lees presented a case demonstrating adrenal insufficiency during surgery.

Dr. Donald MacMillan, assistant at the Jeffrey Hale Hospital, has been granted his Certificate in Anaesthesia from the Province of Quebec.

Dr. J. M. Elliott was the guest speaker at the December meeting of La Société d'Anesthésie de Québec. His subject was: "Ce que les Chirurgiens pensent des Anesthésistes."

Lawrence Corriveau, lawyer, was the guest speaker at the February meeting of La Société d'Anesthésie de Québec. His subject was: "The Legal Aspects of Anaesthesia."

La Société d'Anesthésie de Québec was founded three years ago under the presidency of Dr. Lucien Rinfret, with ten members. There are now forty members. The meetings are held regularly on the first Tuesday of the month at La Maison des Anciens de Laval.

The following changes have been made recently in the staff of the Department of Anaesthesia at the University of Western Ontario Medical School:

Retired: Dr. F. C. Ferguson 31 years' service
Dr. A. S. Graham 28 years' service
Dr. G. W. A. Aitken 21 years' service
Dr. G. A. Wainwright 3 years' service

Appointments:
Dr. H. T. Norry to Victoria Hospital
Dr. Sheilah McLachlin to St. Joseph's Hospital
Dr. Helen Rossiter to St. Joseph's Hospital
Dr. R. F. Ohlke to Westminster Hospital.

Arrangements have been made with University of Western Ontario Medical School for postgraduate study whereby the Resident in Anaesthesia at Victoria Hospital, London, acts as a Fellow part-time in the Department of Physiology and the Senior in Anaesthesia acts as an Instructor part-time in the Department of Pharmacology.

In December the Section on Anaesthesia of the London Academy of Medicine met conjointly with the Society of Obstetricians and Gynecologists of Western Ontario at the London Hunt and Country Club. Speaker Dr. John Mann of Toronto.

Recent successful candidates in the Specialty Examinations, who completed

their postgraduate training under the supervision of the University of British Columbia, Department of Anesthesia, were:

Dr. Jone Chang, F.R.C.P. (Anaesthesia)
Dr. William Albert Dodds
Dr. Lois Dorothy Crawford
Dr. George Edward Sleath
Dr. Doris Elspeth Mackay
Dr. Patrick Nylan Kenny

Dr. George F. Day, who took his postgraduate Anaesthesia training in Montreal and Vancouver, has joined the staff of Associated Anaesthetic Services, and the Anaesthetic Department of the Vancouver General Hospital.

Dr. Howard C. Carruthers, who took his postgraduate training at the Virginia Mason and Childrens Orthopedic Hospital, in Seattle, has joined the staff of Associated Anaesthetic Services, and the Anaesthetic Department of the Vancouver General Hospital.

Dr. Patrick N. Kenny, who completed his Anaesthesia training in Vancouver, has established practice in Chatham, Ontario.

Dr. Horace B. Graves was a recent speaker (on dental anaesthesia) at the Canadian Dental Association.

Dr. H. H. Macartney was a guest speaker at the Clinical Meeting of the Department of Surgery, University of British Columbia. Dr. Macartney spoke on experimental work with cardiac arrest and ventricular fibrillation.

Dr. W. A. Dodds and Dr. J. B. Fulton, of the Department of Anaesthesia, Vancouver General Hospital, and the University of British Columbia, have returned from an extensive tour of Denver, Minneapolis, Philadelphia, and Toronto, where they studied the modern methods and clinical evaluation of "Hypothermia Anaesthesia."

Dr. Arthur E. Guedel, of Los Angeles, spoke at a combined meeting of the B.C. Division, Canadian Anaesthetists' Society, and the regular Staff Meeting of the Department of Anaesthesia, Vancouver General Hospital.

Dr. Elsie Ritch, Anaesthesiologist with the Canadian Army, has joined the Resident Staff of the Vancouver General Hospital, to complete her prescribed training leading to certification in Anesthesia.

Dr. Robert Hugh Thorp, who was recently in the service of the Imperial Oil Limited, as regional physician in the Northwest Territories, has enrolled in the postgraduate Course in Anaesthesia, at the University of British Columbia, and is now on the Resident Staff of the Shaughnessy Veteran's Hospital.

Dr. Angus MacMillan, Dr. Jone Chang, and Dr. Hugh H. Macartney plan to attend the Canadian Anaesthetists' Society Western Division Meeting, and present papers.

Over 19,000 anaesthetics were administered by the Department of Anaesthesia, Vancouver General Hospital, in 1954.

Drs. H. A. Kester, J. C. Poole, E. Webb, R. S. Woodsworth, L. D. Crawford, L. E. Davies and J. T. Hugill plan to attend the meeting of the Western Society of Anesthesiologists in San Francisco, in March, 1955.

Dr. D. Kyle, who has recently completed his Anaesthesia training at the Vancouver General Hospital, has joined the staff of the University Hospital, Edmonton, Alberta.

A series of sixteen lecture demonstrations in general pathology for the anaesthesiologist, has been inaugurated by the Department of Pathology, University of British Columbia.

A Seminar Club for the postgraduate students in Anaesthesia, University of British Columbia, is held every second Wednesday evening at the homes of the staff anaesthesiologists. Recent topics were: "Poliomyelitis"; "Hypothermia"; "Review of Literature on Barbiturate Poisoning."

University of Saskatchewan

The new Department of Anaesthesia at the University of Saskatchewan will begin to function as a clinical unit on January 26, 1955. This has been set as the opening date for the new 550-bed hospital.

So far the following have been or are in the process of being appointed to the staff of the Department:

Dr. Gordon M. Wyant has been appointed Professor of Anaesthesia and Dr. Christopher J. Kilduff of East Grinstead, England, becomes Instructor in Anaesthesia. Drs. Robert S. Daymond and William E. Upthegrove, both of Saskatoon, and Dr. Beverley C. Leech of Regina are to be Visiting Consultants. Dr. Mary V. Morton has been appointed to the active part-time staff; Dr. Glen W. Kinsman of Saskatoon will also be associated with the Department on a part-time basis.

The Department will provide a three-year course of training for residents in Anaesthesia, and the initial number of residents has been set at three. It is expected that residents will be trained both for certification and for the Fellowship examination of the Royal College of Physicians of Canada. An active research programme is planned for the Department and it is hoped that the resident staff will participate in it. The activities of the Department will include, apart from the training of residents, tuition of undergraduates as well as postgraduate courses for part-time anaesthetists in practice in the Province.

BOOK REVIEW

A MANUAL OF REGIONAL ANESTHESIA FOR PRACTITIONERS OF MEDICINE, by JOHN ADRIANI. First edition, Springfield, Ill.: Charles C. Thomas; Toronto Ryerson Press. Price $7.25.

Written by one of the most eminent teachers of anaesthesia in America, this book provides a detailed discussion and description of technique for performing a great variety of diagnostic and therapeutic nerve blocks and blocks for surgical anaesthesia.

The manual is essentially a revision and amplification of the section on regional anaesthesia from Dr. Adriani's *Techniques and Procedures of Anesthesia*, and is arranged in an orderly manner familiar to those who have read that text, and *The Pharmacology of Anesthetic Drugs*.

Excellent illustrations, diagrammatic for the most part, and a few photographs, provide one of the best features of this text-book. Their usefulness is enhanced by their being in the proper place, that is, facing the text to which they apply.

The descriptions are for the most part clear and concise. For the beginner, however, it would seem that one technique would be better than the description of a variety of techniques, such as those given for block of the brachial plexus.

The Preface states that the book has been prepared for those who are beginners in the study of regional anaesthesia, and for those who may need to perform an occasional block. Yet detailed instructions are given for alcohol blocks of certain nerves, notably the divisions of the fifth cranial nerve, and for an intraspinal alcohol technique. This would appear to be inconsistent with the Preface, as these procedures can hardly be considered as suitable for either a beginner or the occasional worker to attempt.

The outline form of this manual, the excellent illustrations, and the logical arrangement of material should make this an exceedingly valuable addition to the library of any anaesthetist.

I. M. K.

WORLD FEDERATION OF SOCIETIES OF ANESTHESIOLOGISTS AND WORLD CONGRESS OF ANESTHESIOLOGISTS

PLANS are progressing favourably for a great international gathering of anaesthesiologists to be held at Scheveningen, near The Hague, Holland, September 5–10, 1955. The Netherlands Society of Anesthesiologists will be our hosts, and they are preparing a scientific programme for the Congress. Societies of anaesthesiologists in twenty-five countries have signified their intention to become members of the new World Federation which has been under consideration by an Organizing Committee for the past four years. The World Congress of Anesthesiologists and the inaugural Assembly of the World Federation are under the high patronage of Her Majesty The Queen of the Netherlands, who has graciously consented to open the proceedings in a formal ceremony to be held at the Parliament Buildings in The Hague.

The Congress will be devoted to discussion of scientific subjects and a large number of papers will be presented by anaesthesiologists from many countries. The official languages of the Congress are English, French, and German; and translation into English will be available at all times. The principal subjects which will comprise the programme of the Congress are:

(*a*) Muscle Relaxation;
(*b*) Hypotension;
(*c*) Hyperthermia and Artificial Hibernation;
(*d*) Respiration and Circulation;
(*e*) The Teaching of Anaesthesia.

All inquiries and communications regarding the scientific programme, registration, hotel accommodation, etc. should be addressed to Mr. W. A. Fentener van Vlissingen, Executive Manager, World Congress of Anesthesiologists, BILTHOVEN, Holland. The Chairman of the Organizing Committee for the Netherlands is Dr. C. R. Ritsema van Eck, Groningen; the Secretary, Dr. M. Mauve, Heemstede; and the Treasurer, Dr. L. A. Boeré, Leiden. Lady B. M. de Beaufort is in charge of the ladies' entertainment and the social aspects of the programme, which will include trips through interesting old towns, showing canals, windmills, flower sales, etc. Registration fee for the Congress will be 35 Dutch guilders (approximately $8.00 Canadian) for active members, and 10 guilders for lady guests.

Last June, I attended a meeting of the Organizing Committee for the World Federation of Societies of Anesthesiologists which was held at Scheveningen, and I had an opportunity to visit the hotels and the meeting-halls which will accommodate the World Congress. There are three resort hotels adjacent to the meeting-hall on a beautiful beach facing the North Sea. Accommodation and service are excellent, and the meals very good indeed—in fact I was very favourably impressed by the whole attitude of our Dutch colleagues and by the excellence of the preparations which they are making for the Congress. The name of Canada is loved and respected in Holland, and I am sure that every Canadian anaesthetist who attends the Congress will have a wonderful time.

The World Federation, which will come into being at the same time as the Congress, will be a continuing body which we hope will bind together anaesthetists in all countries in order to provide better anaesthesia for all the peoples of the world. The Federation will act as a clearing house for information regarding standards of anaesthesia training, available residencies, and other matters of common interest. It will help the organization of societies of anaesthesiologists in countries where the specialty is just beginning, and it will hold periodic World Congresses at intervals of perhaps three or four years. The most important officer of the new Federation will be the Secretary-Treasurer, to be elected by the first General Assembly. The Association of Anaesthetists of Great Britain and Ireland have named Dr. Geoffrey Organe of London for this post, and the members of the Organizing Committee hope that the delegates to the General Assembly will accept this very suitable nomination. The headquarters of the Federation will then be set up, in a modest way, in London.

According to the proposed Constitution of the World Federation, the Canadian Anaesthetists' Society will be entitled to three official delegates to the General Assembly.

The General Assembly will elect an Executive Committee which will carry on all the activities of the Federation in the interval between General Assembly meetings.

I hope that Canada may be well represented in both the Federation and at the World Congress.

HAROLD R. GRIFFITH, M.D.

Montreal, January 13, 1955

WORLD FEDERATION OF SOCIETIES OF ANESTHESIOLOGISTS

(Draft Constitution, as revised June, 1954)

SECTION I. *Name and Headquarters*

Art. 1—In conformity with the decisions of its first General Assembly of 1955, an international non profit making scientific Society to be known as the WORLD FEDERATION OF SOCIETIES OF ANESTHESIOLOGISTS is hereby set up.

Art. 2—The statutes of the Federation shall be registered in. . . .

SECTION II. *Purposes and Functions*

Art. 3—The object of the Federation is to make available the highest standards of anesthesia to all peoples throughout the world.

In pursuit of this aim, the functions of the Federation shall in particular include the following:

(*a*) To assist and encourage the formation of national Societies of anesthesiologists.
(*b*) To promote the dissemination of scientific information.
(*c*) To recommend desirable standards of training for anesthesiologists.
(*d*) To provide information regarding opportunities for postgraduate training and research.
(*e*) To encourage research into all aspects of anesthesiology.
(*f*) To encourage the establishment of safety measures including the standardization of equipment.
(*g*) To advise upon request national and international organizations.

SECTION III. *Membership*

Art. 4—The members of the Federation shall be the national Societies known to and certified by the Interim Committee for the establishment of this Federation including the National Societies of Argentine, Austria, Australia, Belgium, Brazil, Canada, Chile, Colombia, Cuba, Denmark, Finland, France, Germany, Great Britain & Ireland, Israel, Italy, Netherlands, New Zealand, Norway, Philippines, South Africa, Sweden, Switzerland, Spain, United States of America, Uruguay, together with such organizations as shall be approved by a vote of a majority of the aforesaid members present at the first General Assembly of the Federation which shall be held without notice immediately following the adoption of these articles.

Art. 5—Membership of the Federation shall be granted to other national organizations approved by the General Assembly in accordance with these articles and following the procedure determined in the by-laws.

Art. 6—Only one organization per country shall be permitted to membership of the Federation.

Art. 7—In addition to member organizations there shall be "Associates of the Federation" consisting of anesthesiologists holding a registered medical qualification from countries where there is no recognized organization of anesthesiologists. Associates will be admitted by the Executive Committee after application and shall have such rights, privileges and duties and shall pay such annual subscription as the Executive Committee may determine. The Executive Committee shall have the right at any time to suspend the membership of an Associate of the Federation who shall have the right of appeal to the next General Assembly. The General Assembly may by a two-thirds majority expel such Associate member.

Art. 8—A member organization or an "Associate member" may resign at any time following the procedure determined in the by-laws. Membership of a member organization may be terminated by a vote of the General Assembly by a two-thirds majority in the following cases:

(*a*) if a change in the nature of the activities of the member organization makes it inappropriate for continued membership;
(*b*) if the member organization wilfully defaults in the payment of its subscription.

Art. 9—Organizations which for any reason cease to be members of the Federation shall have no rights in the Federation's funds and shall not be entitled to demand a statement of account, to affix the seals to the Federation's property nor to demand an inventory.

SECTION IV. *General Assembly*

Art. 10—The Federation shall be governed by a General Assembly of delegates from member organizations. Each member organization shall be entitled to one delegate if the number of its members is 250 or fewer. It will be entitled to 2 delegates if it numbers from 251 to 500 members; to 3 if it numbers from 501 to 1,000 members, and to one extra delegate per 1,000 members over 1,000. The delegates must be qualified anesthesiologists.

Any national member organization which desires to ensure its full quota of representatives at meetings of the General Assembly by the appointment of alternates for its regularly named delegates may do so, provided that no such alternate shall be permitted to participate in the work of the General Assembly until he has been duly certified to the Secretary-Treasurer of the Federation as assuming the powers and duties of the delegate he has been designated to replace.

No person shall be a member of the delegation of more than one country.

Art. 11—Voting by the General Assembly shall be by individual delegates.

Art. 12—Each member organization shall be required to forward to the Secretary-Treasurer the name(s) of its accredited representative(s) before the opening of the session of the General Assembly.

Art. 13—The General Assembly shall meet in ordinary session on the occasion of each international congress. It shall meet in extraordinary session as circumstances may require.

Extraordinary meetings of the General Assembly shall be convened by the Executive Committee, either on its own responsibility or at the request of not less than two-thirds of the member organizations.

Art. 14—The General Assembly shall be the supreme body of the Council and shall be entitled to take cognizance of all matters not explicitly precluded by these articles.

The principal functions of the Assembly shall be:

(*a*) to determine the policy of the Federation;
(*b*) to elect the Executive Committee and the Secretary-Treasurer in accordance with these articles;
(*c*) to receive and approve the reports of the Executive Committee;
(*d*) to fix the scale of subscriptions, to supervise the financial policy of the Federation and to scrutinize and approve the budget and accounts.

Art. 15—The Executive Committee shall prepare the agenda of the Assembly and shall forward the same to full members simultaneously with the notice of the Assembly.

Art. 16—The General Assembly shall elect a president at the beginning of each session with three vice-presidents or more at its discretion.

The Assembly shall deal with the items on the Agenda and such other matters as it may decide to add thereto.

The Assembly shall adopt its rule of procedure.

Art. 17—Decision shall be by simple majority of the members present and voting, affirmative or negative votes alone being valid; nevertheless a majority of two-thirds shall be necessary for the admission or expulsion of members or for the amendment of these statutes.

The Assembly shall only be entitled to debate such amendments if they have been included in the agenda attached to the notice of the meeting, or if not less than two-thirds of the full members are present or represented.

If a vote is equally divided, the president shall have a casting vote.

Art. 18—Decisions of the General Assembly shall be recorded in minutes signed by the president and the executive secretary and by such members as desire to do so. The Minutes shall be kept at the headquarters of the Federation and shall be open for perusal by all interested parties. Copies or extracts from the minutes shall be signed by the chairman of the Executive Committee and the Secretary-Treasurer.

One free copy of all minutes shall be forwarded by the Executive Committee or the Secretariat to each full member.

SECTION V. *Executive Committee*

Art. 19—The Executive Committee shall execute the decisions of the General Assembly and take within the limits of those decisions all measures designed to further the purposes of the Federation.

The Executive Committee shall consist of 12 members of whom at least one must be from (*a*) Europe, (*b*) the U.S.A. and Canada, (*c*) Latin America, (*d*) Asia and Australasia, and one member from each of the two largest member organizations.

The Executive Committee shall have power to co-opt to three additional members who shall serve until the next period of reelection.

Half the members (other than the Secretary-Treasurer) of the Executive Committee shall retire at each regular meeting of the General Assembly and shall not be eligible for immediate reelection.

Outgoing members in the first instance shall be selected by lot, and subsequently by rotation.

Where vacancies arise from retirement or other causes, the Executive Committee itself shall fill such vacancy or vacancies provisionally.

Persons elected to the vacancies aforesaid shall serve for the remainder of the term of the member they replace. Where requisite, the next General Assembly shall proceed to a regular election.

Art. 20—The Chairman and his deputies will be appointed by the Executive Committee from among its members.

The Secretary-Treasurer, who will have right to vote, shall be elected by the General Assembly.

The Executive Committee shall nominate the Secretary-Treasurer three months in advance of the General Assembly.

Counter nominations may be made by three voting members of the General Assembly, nominations to be received at least two months before the General Assembly.

The Secretary-Treasurer shall hold office until the termination of each regular meeting of the General Assembly and shall be available for immediate reelection.

Art. 21—The Executive Committee shall meet before and during each session of the General Assembly and whenever the interests of the Federation so requires on the summons of its chairman or his deputy.

Art. 22—Voting in the Executive Committee shall conform to the rules laid down in these articles for voting in the General Assembly.

The Secretary-Treasurer may request the Executive Committee to take decisions by postal ballot.

Art. 23—The Secretary-Treasurer shall forward to each member organization an annual report of the Federation.

Section VI. *Finance*

Art. 24—The resources of the Federation will originate from:

(*a*) Annual contribution paid by member organizations, the scale of which shall be fixed by the General Assembly and shall be related to the number of votes of each member association:

(*b*) Annual subscription of the associates of the Federation:

(*c*) Subsidies, gifts and bequests that might be bestowed on the Federation and accepted by the Executive Committee:

(*d*) Profits accruing from international congresses.

Art. 25—The Secretary-Treasurer shall make up the accounts and budgets of the Federation as at 31 December yearly and prepare the same for inspection by 31 March following at the latest. The audited accounts shall be forwarded to member organizations together with the yearly report.

Section VII. *Dissolution and Liquidation*

Art. 26—In the event of the dissolution of the Federation for any reason or at any time, liquidation shall be carried out by the person appointed by the Genneral Assembly, which shall decide upon his powers and, if relevant, his emoluments.

Failing the appointment of a liquidator, liquidation shall be carried out by the Executive Committee then in office acting as a Panel of Liquidators. Such Panel shall have powers without restrictions or limitations.

SECTION VIII. *Entry into Force*

Art. 27—These statutes shall become effective when ten of the organizations participating in the first General Assembly shall have accepted them.

The several governing or administrative organs of the Federation shall then take up their full duties in accordance with the directives received.

Art. 28—The English text of the statutes and by-laws shall be considered as authoritative.

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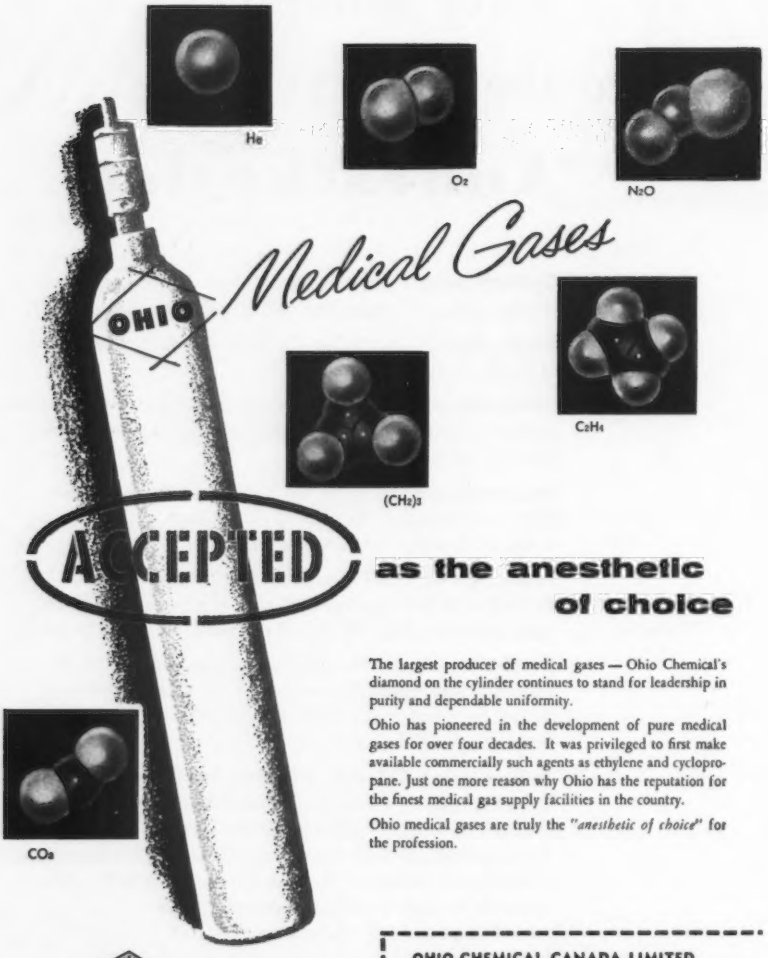

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1. Heinberg, C.J.: Eye, Ear, Nose & Throat Monthly 30:31 (Jan.) 1951.

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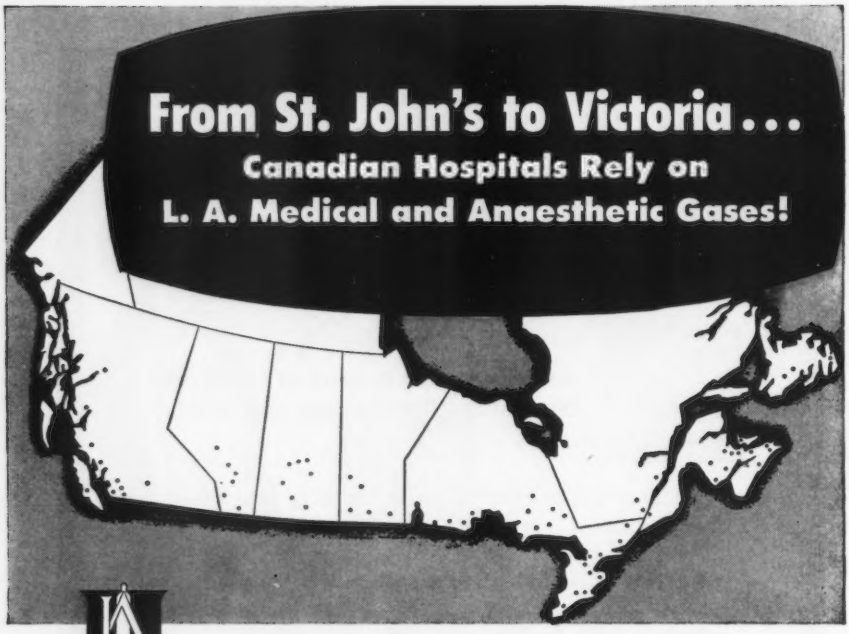

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References:

1) C. Thelen, **Jackson Clin. Bulletin 12**:119, 1950.
2) W. M. Kane, **Am. J. Obst. & Gynec., 65**:1020, 1953.
3) L. E. McCrea and E. Post, **J. of Internat. College of Surgeons, 21.4**, 1954.
4) H .W. Gierson, L. S. Gottlieb, H. J. Rubin, **Dis. of the Chest, 27**:1, Jan. 1955.

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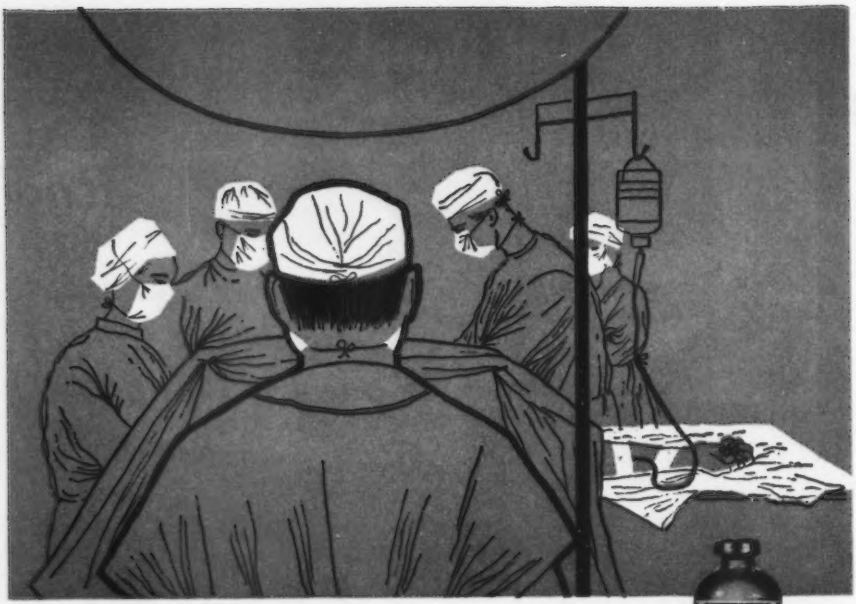

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1. Helrich, M.; Papper, E. M., & Rovenstine, E. A.: *Anesthesiology* 11:33, 1950. 2. Stephen, C. R., & Martin, R.: *North Carolina M. J.* 12:501, 1951. 3. Phillips, H. S.: *Anesth. & Analg.* 32:56, 1953.

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Articles should be typewritten in double space on one side of the paper only. Pages must be serially numbered, and each page should carry at its head the name of the author and the title of the article in full or in an appropriate abbreviation. The article should be concluded by a summary which will be intelligible without reference to the main text. All articles should be accompanied by a résumé presenting the important features in short form, for translation into the French language. French-speaking authors should provide this résumé in the French language.

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4. Griffith, H. R. & Johnston, G. E. The Use of Curare in General Anaesthesia. Anesthesiology 3: 481 (1942).

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Labat, G. Regional Anesthesia. 1st ed., Philadelphia: Saunders (1922).

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